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IRIDENCLEISIS CUM IRIDOTOMIA MERIDIONALI

AN OPERATIVE METHOD BOTH IN ACUTE AND IN CHRONIC PRIMARY GLAUCOMA *

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Dr. Arnold Knapp, who has tried my extralimbal, tangential punch forceps sclerectomy for chronic primary glaucoma,[1] wrote to me in 1927, after the appearance of my paper on iridencleisis in acute glaucoma,[2] that he was somewhat puzzled by my papers, particularly by the fact that I had abandoned one method for another.

From my list of publications [3] it will be easily understood that Dr. Knapp is right, especially when I state that among 640 operations for primary chronic glaucoma carried out in my private practice during

* Submitted for publication, Sept. 16, 1930.

* Read at the discussion on the operative treatment for glaucoma at the Eighth Scandinavian Ophthalmological Congress, Helsingfors, Finland, June, 1930.

1. Holth: A New Technic in Punch Forceps Sclerectomy for Chronic Glaucoma: Tangential and Extralimbal; Iridencleisis Operations Epitomized 1915-1919, Brit. J. Ophth. **5**:544, 1921; also read at the Oxford Ophthalmological Congress, July 16, 1920; On the Technique in Extralimbal Tangential Punch Forceps Sclerectomy for Chronic Glaucoma, Acta ophth. **3**:62, 1925; also read at the Sixth Scandinavian Ophthalmological Congress, Copenhagen, June 29, 1925.

2. Holth: Iridencleisis cum iridotomia meridionali bei Glaucoma acutum, Klin. Monatsbl. f. Augenh. **79**:620, 1927.

3. Holth: Discussion sur la sclérecto-iridectomie de Lagrange, Bull. et mém. Soc. franç. d'opht. **24**:492, 1906; Ein neues Prinzip der operativen Behandlung des Glaukoms (Fistula subconjunctivalis camerae anterioris), Ber. d. ophth. Gesellsch., Heidelberg, 1906, p. 123; Iridencleisis antiglaucomatosa, Ann. d'ocul. **137**:345, 1907; De l'action favorable de l'iridencleisis dans le cas de glaucome chronique ou l'iridectomie et la sclérotomie antérieure sont restées insuffissantes, XI. Cong. internaz. di oftal., Napoli **1**:245, 1909; Sclérectomie antérieure à l'emportepièce dans le glaucome, de préférence après incision à la pique, Bull. et mém. Soc. franç. d'opht. **27**:326, 1909; also Ann. d'ocul. **142**:1, 1909; On My Technique in Limbal Sclerectomy for Glaucoma, Ophthalmoscope **9**:487, 1911; also read at the Oxford Ophthalmological Congress, July, 1910; Anatomische Untersuchung der Operationsnarben und der aplanierten Papillenexkavation nach erfolgreichen Glaukomoperationen (Iridencleisis, Sclerectomia limbalis nach Holth oder Elliot, Sclerecto-Iridencleisis), Ber. d. ophth. Gesellsch., Heidelberg, 1913, p. 355; footnote 1; Anatomical Examination of 6 New Cases of Subconjunctival Fistula Scars from 5 Months to 6 Years after Successful Iridencleisis or Limbal Sclerectomies in Chronic Glaucoma, Brit. J. Ophth. **6**:10, 1922; also read at the Fifth Scandinavian Ophthalmological Congress, Stockholm, Aug. 28, 1921; footnote 2.

25 years (from 1904 to 1929), 223 were iridencleises, 268 punch forceps sclerectomies and 149 trephinings (Elliot). Iridectomies that I had carried out for primary acute glaucoma before 1916, as well as those for secondary, and above all for uveitic glaucoma, have not been counted. In two early papers [4] I mentioned my predecessors in iridencleisis for glaucoma: Bader and Herbert.

After iridencleisis and punch forceps sclerectomy, as well as after trephining, I have often obtained permanent tonometrically normal tension without miotics and with visual fields and visual acuities preserved unchanged for years. I have had the opportunity of making microscopic examinations of subconjunctival fistulas [5] post mortem up to six years after successful operation. Further, I have been able to demonstrate microscopically the aplanation of the glaucomatous excavations in the optic disk, the depth of which was measured before operation by ophthalmoscopy in direct image.[6]

The twenty-two patients whom I demonstrated on June 27, 1907, at the third Scandinavian Ophthalmological Congress at Christiania (Oslo),[7] showing tonometrically normal tension obtained in thirty eyes without miotics after iridencleisis for primary chronic glaucoma, are now all dead. A woman, aged 77, who was operated on in October, 1907, for chronic glaucoma in both eyes is still alive. After iridencleisis she had, when last observed, tonometrically normal tension, without the use of miotics and well conserved visual fields in both eyes. On account of a senile cataract that had developed in recent years, her right eye had a vision of 1/20 in June, 1930, while the left eye had a vision of 5/15. She was able to read papers and books easily with the help of weak convex glasses.

I shall now state briefly why I have abandoned the three methods of anterior sclerectomy that I have tried: my first punch forceps operation, Elliot's trephining and the tangential punch forceps sclerectomy. When the anterior chamber was shallow, the last mentioned procedure could only be limbal—never extralimbal. All three methods give an immediate, sometimes brutal, fall of tension. After several years, however, the defect in the sclera may be completely obliterated by solid and impermeable scar tissue. The tension is then often raised again.

Three years after a successful punch forceps sclerectomy on one eye and after a successful Elliot operation on the other, I showed microscopically post mortem this solid scar tissue developing from the sclera as well as from the subconjunctival scar tissue.[8] If the patient had lived more than three and one-third years after operation, the

4. Holth (footnote 3, second and third references).
5. Holth (footnote 3, seventh and ninth references).
6. Holth (footnote 3, seventh reference).
7. Holth: Norsk mag. f. Lægevidensk. **6**:263, 1908.
8. Holth (footnote 3, seventh reference, figs. 5, 6 and 7).

scleral defects of the two eyes would probably have been closed by impermeable scar tissue in the center.

After my iridencleisis the condition is then reversed: In only 50 per cent does the tension become at once and permanently normal. In 35 per cent one must use miotics from a fortnight to half a year after operation to keep the tension normal. In both of these instances the resulting "filtering scar" will nearly always last for a lifetime. In the remaining 15 per cent a continued use of miotics, which are useless before operation, will keep the tension normal in most cases and as a rule reoperation can be avoided.

Morax [9] reported in 1921 that, according to his experiences in 426 cases in private practice, anterior sclerectomy was not superior to iridectomy. Marbaix [10] found the development of "filtering scars" only when the iris was included in the incision scar; in 1909, I said [11] that this inclusion probably often occurred in Lagrange's sclerecto-iridectomy.

As regards the dreaded late infection, I have seen two cases of panophthalmitis after Elliot's operation and one case after my punch forceps sclerectomy of 1909. In the last mentioned case the infection occurred in the patient's best eye, which for seven years had had normal tension, a good visual field and a vision of 5/4. Besides these, I have seen several other cases of late infection following both operations. These infections, however, were localized at the scar until the infection was stopped by the instillation of ethylhydrocuprein (1 per cent) several times a day and of silver nitrate (0.5 per cent) once a day; thus the functions of the eye were unhurt. I have also seen the same occurrence in two instances after my tangential punch forceps sclerectomy.[1] In both cases the anterior chamber was shallow, the sclerectomy was accordingly limbal, and not extralimbal, and thus the raison d'être of the operation diminished.

After iridencleisis in seeing eyes I have never observed any late infection. In one case of glaucoma absolutum dolorosum, however, in which the patient had refused the proposed enucleation, I tried iridencleisis; a late infection manifested itself two years after the operation with panophthalmitis, and exenteration of the bulb was performed. In another case of glaucoma absolutum dolorosum (in a patient with syphilis and dipsomania) a superficial infection was seen nine years after the operation, but disappeared in a few days owing to the instillation of silver nitrate (0.5 per cent) once a day; the patient lived for several years with normal tension and absence of pain. The reason

9. Morax, quoted by Peters, A.: Das Glaukom, Berlin, Julius Springer, 1930. (This book contains references to the literature on glaucoma from 1908 to Jan. 1, 1930.)

10. Marbaix, quoted by Peters: Das Glaukom.

11. Holth (footnote 3, fifth reference).

why the late infection occurs so seldom after iridencleisis is to be found in the fact that the subconjunctival end of the iris fistula always is placed further away from the limbus than the sclerectomy fistula, even if the anterior chamber is shallow (figs. 2 and 3).

Dr. Enroth [12] has shown that cataract may occur relatively early after Elliot's operation. I must own that I have not undertaken a systematic investigation of this possibility, either after trephining or after my two punch forceps sclerectomies. In a man, aged 66, who had chronic glaucoma in both eyes, with a few thin radial opacities of the periphery of the lens, a cataract developed within three years after a successful tangential sclerectomy performed in November, 1920. The lens of the other eye, the tension of which was kept normal by the instillation of pilocarpine (2 per cent) twice a day, remained unaltered. When last observed, the patient, at 76 years of age, still had a good visual field and a vision of 5/7.5, as he had had ten years before.

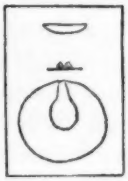

Fig. 1.—Iridencleisis cum iridotomia meridionali with the stop keratome; used in cases of chronic primary glaucoma with normal depth of the anterior chamber. The subconjunctival keratome incision, 6 mm. long, is made from 1.5 to 2 mm. from the corneal limbus.

I have never observed this result following iridencleisis cum iridotomia meridionali, except when the operation had to be performed in far advanced cases of cataract. When I must perform a cataract extraction in such cases, I first make Liégard's preliminary suture upward near the limbus and then a radial clip in the lower half of the iris (before the cystotomy) with the two blunt blades of de Wecker's scissors introduced in the anterior chamber.

The partisans of cyclodialysis—especially those who consider causing atrophy of the cillary body to induce a fall of tension—should think of the possibility of cataract caused by that operation.

As long ago as in 1906 I demonstrated microscopically a subconjunctival fistula of the iris as the cause of a "filtering scar" following an iridencleisis cum iridotomia meridionali.[13]

12. Enroth, Emil: Ueber Katarakt nach Elliot's Operation, Acta ophth. **5**:112, 1927.

13. Holth (footnote 3, second reference, fig. 3).

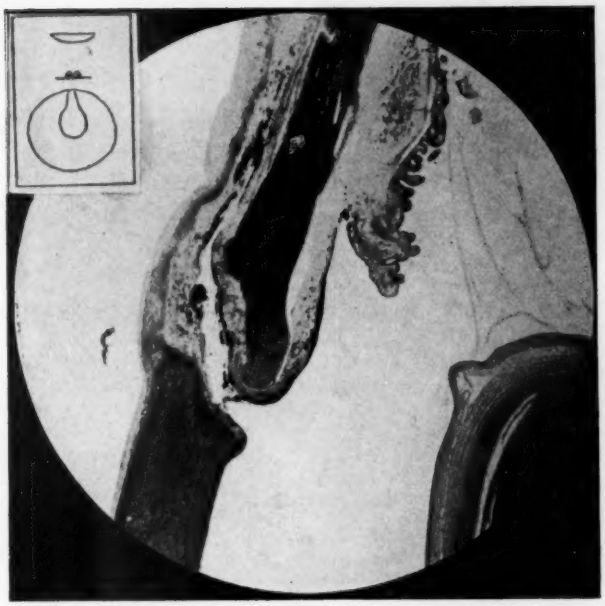

Fig. 2.—Axial vertical section through subconjunctival iris fistula of the right eye after iridencleisis cum iridotomia meridionali performed four and one-half years before death (Holth: Ber. d. ophth. Gesellsch., Heidelberg, 1913, p. 355, fig. 1); × 18. This photomicrograph and those shown in figures 7, 8, 9 and 11 were made by Prosector Dr. Ole Berner.

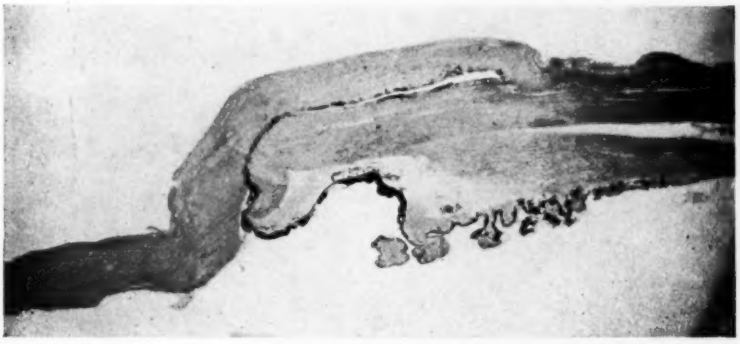

Fig. 3.—Axial vertical section through subconjunctival iris fistula of the left eye after iridencleisis cum iridotomia meridionali, performed five months before death (Holth: Brit. J. Ophth. **6**:10, 1922, fig. 2); × 15.

Wessely [14] considered this rather an exception and held the opinion, based on experiments on animals, that it is not conclusive that the iridencleisis is responsible for the fall of tension after a fistula-forming operation. In 1907, however, I called attention to the fact that I had performed subconjunctival iridencleisis in different ways in rabbits (from May to August, 1904) and always had failed in obtaining a "filtering scar" as in man.[15] After this failure I performed a voluntary subconjunctival iridencleisis in August, 1904, on a woman with glaucoma absolutum; the patient obtained a "filtering scar" and normal

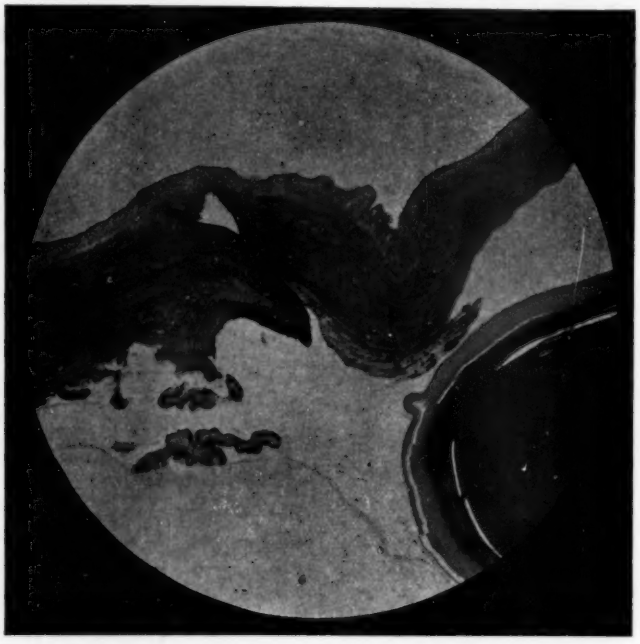

Fig. 4.—Axial vertical section through subconjunctival iris fistula of the right eye after iridencleisis cum iridotomia meridionali, performed six years before death (Holth: Brit. J. Ophth. **6**:10, 1922, fig. 4); × 18.

tension. Later,[16] I was able to show post mortem, from five months to six years after subconjunctival iridencleisis in [17] seeing eyes (figs. 2, 3, 4 and 5), that a subconjunctival iris fistula was the cause of the "filtering scars" obtained and thus of the normal tension and the good

14. Wessely, quoted by Peters: Das Glaukom, p. 332.
15. Holth (footnote 3, third reference).
16. Holth (footnote 3, seventh and ninth references)
17. Holth (footnote 3, fourth reference).

functional result; in a fifth case (fig. 7) the same fistula was the cause of a "cystoid scar" and the same result.

Borthen [18] did not believe in the subconjunctival iris fistula and explained the effect of his "iridotasis" as a stretching of the iris upward, thus opening the corneo-iridic angle downward (the superior angle will be almost closed). I disapprove of this "iridotasis" in the normally functioning sphincter iridis in seeing eyes, because when the effect of atropine is over, the pupil becomes eccentric, which is a poor optical result; it is dislocated near the limbus.

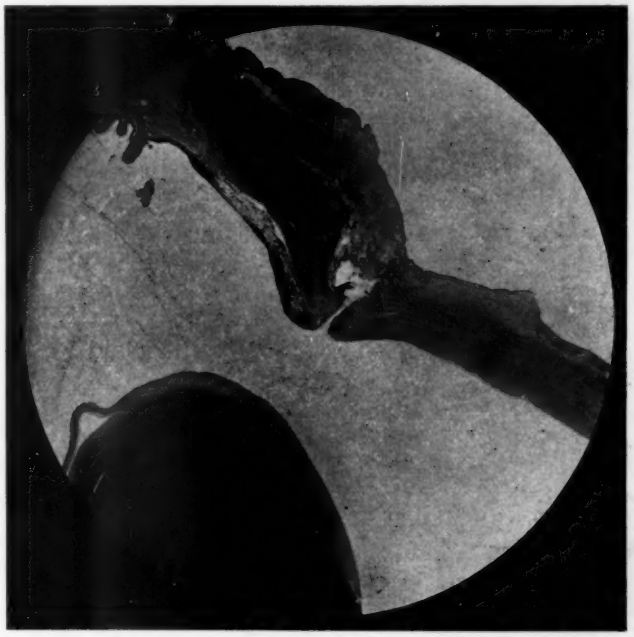

Fig. 5.—Axial vertical section through subconjunctival iris fistula of the left eye (same case as in fig. 8) after iridencleisis cum iridotomia meridionali, performed six years before death (Holth: Brit. J. Ophth. **6**:10, 1922, fig. 3); × 18.

In my meridional iridotomy (fig. 1) the clip through the sphincter iridis causes the pupil to withdraw so much that it becomes nearly central. When the clip is made in the middle of the iridial tent, which is made possible by the removal of the iris forceps immediately before the clip (fig. 15), both sphincter angles retract somewhat into the anterior chamber, but not to their original place.

18. Borthen, Johan: Iridotasis antiglaucomatosa, Arch. f. Augenh. **68**:145, 1910-1911.

Besides iridencleisis cum iridotomia meridionali, which is my main operation, I earlier used [19] other methods of iridencleisis. In one of these a small basal flap of the iris ("iridotomia lobularis") was drawn into the subconjunctival limbal incision together with the adjacent parts of the periphery of the iris. The latter were, however, often drawn into the chamber again by the intact sphincter iridis, and the development of a subconjunctival fistula of the iris was thus made impossible.

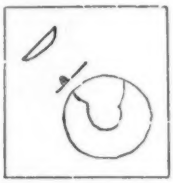

Fig. 6.—Iridencleisis simplex; this is used only when an unsuccessful iridectomy has previously been performed.

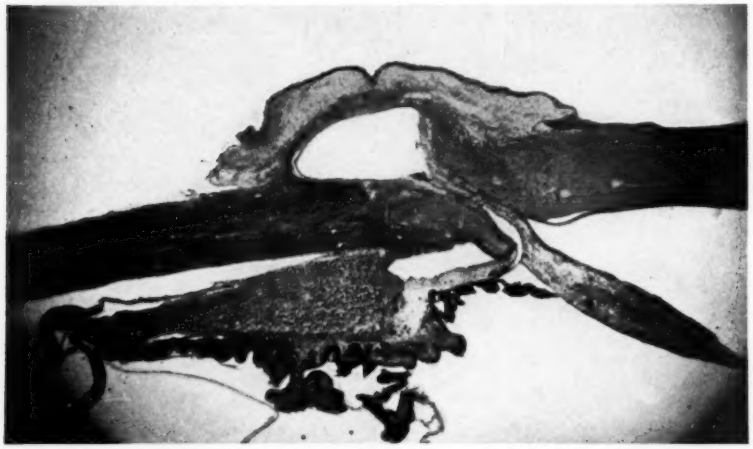

Fig. 7.—Axial vertical section through subconjunctival iris fistula of the left eye after iridencleisis simplex (fig. 10), performed four years and two months before death (Holth: Brit. J. Ophth. **6:**10, 1922, fig. 1); × 18.

I therefore abandoned this method early, but it was the one principally employed by Borthen [18] and Grönholm.[20]

Since April, 1916, I have performed iridencleisis cum iridotomia meridionali in six cases of glaucoma acutum, a rare disease in Norway,

19. Holth (foonote 3, second and third references).

20. Grönholm, quoted by Galetski-Olin, Hanna: Erfahrungen über Iridencleisis, Iridotasis, Sklerektomie und Trepanation, Klin. Monatsbl. f. Augenh. **68:** 139, 1922.

and obtained the best result without any use of miotics. In four of these cases typical "filtering scars" developed; in two cases none was seen. As the anterior chamber here is shallow, the incision must be made near the limbus and be only 4 mm. long (fig. 8) to avoid every possibility of hurting the anterior capsule of the lens or the posterior side of the cornea. The illumination must here, as always, be a sharp electric side light. The presbyopic surgeon ought to use the "Rectavist-Lupenbrille" (legend, fig. 15). I did not make use of general anesthesia in the six cases mentioned; besides the instillation of phenacaine (holocaine, 1 per cent), I injected temporally downward through the lower lid 2 cc. of procaine hydrochloride (4 per cent) with 2 drops of

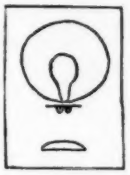

Fig. 8.—Iridencleisis cum iridotomia meridionali; used in cases of a very shallow anterior chamber, as in glaucoma acutum; the subconjunctival keratome incision, only 4 mm. in length, should be performed 0.5 mm. from the limbus corneae.

Fig. 9.—Iridencleisis cum iridotomia transversali (Holth: Klin. Monatsbl. f. Augenh. **79**:620, 1927, fig. 3); proposed for children with hydrophthalmos. This is performed below the cornea under general anesthesia.

epinephrine retrobulbarly according to the method of Duverger;[21] van Lint's[22] akinesia of the sphincter palpebrarum was also performed with the injection of 2 cc. of procaine hydrochloride with 1 drop of epinephrine. The six operations were completely free from pain; the patients did not move during the meridional iridotomy (fig. 15). Also in chronic glaucoma I often use van Lint's akinesia and Duverger's retrobulbar injection of 2 cc. of procaine hydrochloride; in both methods

21. Duverger, C.: L'anesthésie locale en ophtalmologie, Paris, Masson & Cie, 1920, p. 59, fig. 7.

22. van Lint: Paralysie palpébrale temporaire provoquée dans l'opération de la cataracte, Ann. d'ocul. **1**:420, 1914.

I am satisfied, however, with a 2 per cent solution of procaine hydrochloride.[23] I have never seen intoxication after these injections or any noxious effect in the eye that was not operated on.

In 1927, I proposed [2] for hydrophthalmos an iridencleisis cum iridotomia meridionali downward (fig. 9). In November, 1928, I had no occasion to use this method in a man, aged 32, as another oculist had made a large basal iridectomy downward during the patient's childhood,

Fig. 10.—Dr. Holth's stop keratome, 6 by 6 mm., bent at an angle of 45 degrees. (This and the instruments shown in figures 2, 3 and 4 are obtainable from John Weiss & Son, Ltd., 287 Oxford Street, London, W.)

Fig. 11.—Dr. Holth's blunt double conjunctival hook; this may be obtained in stainless steel.

Fig. 12.—Dr. Holth's fixation forceps, elbowed, with round-ended teeth and without catch; obtainable in stainless steel. This instrument is useful for fixation of the inferior limbus, when the eye has to be turned downward far enough to make the subconjunctival incision near the superior limbus easily with the stop keratome (legend, fig. 14).

Fig. 13.—Dr. Holth's iris forceps, with regulating screw; may be ordered in stainless steel.

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23. Thus I inject, at the most, 3 drops of epinephrine. H. Gjessing, who recently enucleated an eye with glaucoma absolutum haemorrhagicum dolorosum, which had previously not been operated on, used 10 drops of epinephrine added to 5 cc. of procaine hydrochloride (2 per cent) and antipyrine (1 per cent). Immediately after the cutting of the optic nerve the patient had a complete but transitory amaurosis in the other glaucomatous eye. Gjessing explained this as the result of shock or the extraordinary dose of epinephrine (Gjessing, H.: Acta ophth. **8**:206, 1930).

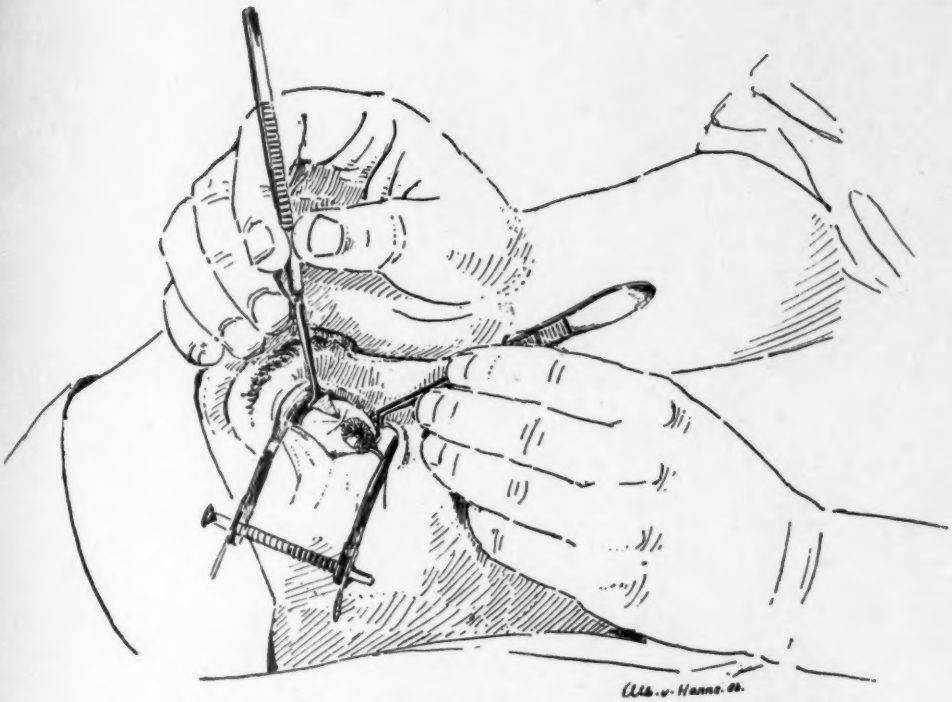

Fig. 14.—Subconjunctival tunnel made with the stop keratome simultaneously with the limbal incision into the anterior chamber. When operating upward (as in most cases), on either eye, the surgeon stands before the patient on his left side; sharp electric illumination is supplied from the patient's right side. The surgeon holds the keratome's handle with his bent, slightly supinated right hand, guiding the point toward himself. The point splits the bulbar conjunctiva, 8 mm. from the corneal border. By very short transverse zigzag movements of the keratome's point the conjunctival fold is slid over the sclera toward the limbus. If the anterior chamber is of normal depth, the scleral incision is made from 1.5 to 2 mm. from the corneal limbus (fig. 5) and the keratome's blade is guided parallel with the iris plane until checked by the knob; then the length of both the conjunctival and the limbal incisions is exactly 6 mm., which is the width of the subconjunctival tunnel connecting the two incisions. In removing the keratome the surgeon has free movement of the hand for lowering the handle and then turning the keratome's point sideways in order to avoid hurting the lens capsule. When the anterior chamber is shallow, as in glaucoma acutum, the incision into the chamber is made 0.5 mm. outside the limbus and the keratome is introduced only 4 mm. into the chamber (fig. 12). For all subconjunctival glaucoma operations the blades of the eye speculum ought to be short in the transverse direction, like the Mellinger speculum in this illustration and in figure 15. By this means the palpebral fissure can be opened well in a vertical direction and there will be plenty of room for making a conjunctival flap with scissors or a subconjunctival tunnel with the keratome.

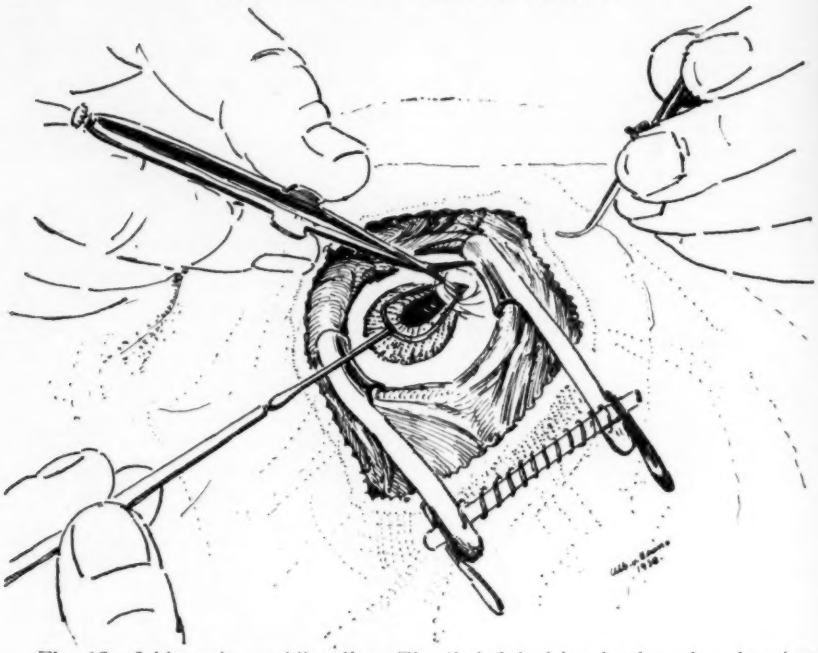

Fig. 15.—Iridotomia meridionalis: The limbal incision in the sclera has just been made with the "stop keratome" through the "subconjunctival tunnel" (fig. 14). The operating surgeon is still standing before the patient but now on his right side, even when operating on the left eye (as shown here); the sharp electric illumination comes from the patient's left side. The assistant, who stands on the opposite side of the surgeon, draws the part of the conjunctival membrane covering the subconjunctival tunnel down to the limbus corneae; in doing this, he makes use of the blunt double hook (fig. 2). The surgeon uses the iris forceps with a regulating screw (fig. 4) which widens it 2 mm.; with this instrument in his left hand he grasps the iris near the edge of the pupil and draws the iris out so far that the upper part of the pupil and, through this, the pigment epithelium on the back of the iridial tent are seen just outside the limbal incision. A presbyopic surgeon should use a "Rectavist-Lupenbrille," with a magnifying power of 1½ for the operating distance of 16.5 cm. (obtainable from Nitsche and Günther-Busch, Rathenow, Germany). Through the extralimbal part of the pupil the surgeon inserts the one blunt blade of de Wecker's iris scissors underneath the back of the iridial tent, while the other blade rests on the front of the tent; he holds the scissors in his slightly supinated right hand when operating on either eye. He now releases the iris from the forceps (the iridial tent lies between the two blades of the scissors) and removes the iris forceps altogether from the field of operation. This is the stage seen in this illustration. The meridional clip through the iridial tent is now made; the two sphincter angles of the narrow coloboma caused by the iridotomia retract somewhat into the anterior chamber; the double hook is removed, and the conjunctival wound is closed by a fine silk thread, which is removed the third day after operation. The open wound treatment is used, although under a perforated aluminum shield. A binocular bandage is superfluous, as the cut through the sphincter pupillae hinders the retraction of the two sphincter angles to their original position.

and later had performed Elliot's operation in vain. I therefore performed my iridencleisis cum iridotomia meridionali upward, using the aforementioned akinesia and retrobulbar anesthesia. I had, however, also paralyzed the rectus superior with the injection of a few drops of procaine hydrochloride (4 per cent) with a little epinephrine. The operation was performed successfully without rupture of the zonula zinnii or loss of vitreous. The tension became normal and a "cystoid scar" developed as shown in figure 7. I do not know whether or not an injection of procaine hydrochloride in the rectus superior will help to prevent the rotation of the eye upward under the general anesthesia necessary in children with hydrophthalmos. If it helps, subconjunctival iridencleisis downward (fig. 9) may possibly be unnecessary. A cystoid scar is disfiguring when it extends downward and may be dangerous in a late infection.

TECHNIC

I recommend the four special instruments shown in figures 10 to 13.

Both blades in the de Wecker's iris scissors must have blunt points, and both blades of the eye speculum (e.g., that of Mellinger, figs. 14 and 15) must be very short in the transverse direction. A fine-toothed forceps facilitates the placing of the double blunt conjunctival hook (fig. 11), and a fine anatomic forceps is handy in making a single suture in the conjunctival incision. The needle-holder, containing a fine needle threaded with black silk thread no. 000, must have a slack catch. All the instruments should be placed in a metal case and at least one day before the operation be sterilized for thirty minutes in dry heat (155 C. or 311 F.). During the operation the instruments are placed separately on a sterilized towel; when an instrument is used a second time, it should first be rinsed in sterilized water.

I prefer the limbal incision (figs. 1, 6, 8 and 9) through the subconjunctival tunnel made simultaneously with the stop keratome (fig. 14), because this method usually produces a "filtering scar" in the form of a flat cushion of conjunctival edema, as over the iris fistulas shown in figures 2, 3, 4 and 5. A subconjunctival "cystoid scar" (fig. 7), which as a rule establishes normal tension, is seen only exceptionally. The subconjunctival tunnel mentioned requires an assistant to draw the conjunctival incision down to the limbus under the meridional iridotomy (fig. 15). The instrument used by the assistant is the blunt double conjunctival hook (fig. 11). One may, of course, dispense with my conjunctival hook, if one makes a short limbal incision and simultaneously an extralimbal conjunctival flap with a narrow von Graefe's cataract knife, as I did some time before 1907. I soon dropped this method because I could not forget an expulsive hemorrhage that occurred twelve hours after a correctly performed iridectomy for chronic glaucoma, in which I had assisted in the spring of 1898. I have never seen an expulsive hemorrhage after a limbal incision with the stop keratome (fig. 10). I have never tried Lundsgaard's[24] limbal incision made with a knife from the

24. Lundsgaard, K. K. K.: On the Indications for a Corneal Section from the Outside with a Knife and the Technique of the Operation, Acta ophth. **6**:311, 1928; also read at the Seventh Scandinavian Ophthalmological Congress, Oslo, June 28, 1928.

outside, because I dared not make Dupuy-Dutemps's detachment of the conjunctiva at the limbus. I know, however, that Lundsgaard now uses a common conjunctival flap, and that he is well satisfied with his results.

When using the stop keratome (fig. 10), one can dispense with the double conjunctival hook (fig. 11) if one omits my subconjunctival tunnel (fig. 14) and in its place makes an ordinary, not too large, conjunctival flap with a pair of scissors beforehand, and after this performs the limbal keratome incision. This was done in the Second Eye Clinic of the University in Vienna,[25] although with the omission of the suture of the conjunctival flap; in my opinion, this suture is absolutely necessary in this procedure. Gjessing's [26] reading of his paper introduced my iridencleisis in Vienna. When he started to perform operations for glaucoma he used Elliot's trephining; since then, in a number of papers,[27] he has demonstrated the advantages of iridencleisis through the subconjunctival tunnel, a procedure of which he is a convinced adherent.

The Seidel fluorescein test, which I perform *before* the anesthesia for tonometry, nearly always is negative in "filtration scars" following my iridencleisis operations; accordingly, these scars almost never show free filtration. Only twice have I seen a positive fluorescein test after iridencleisis, in one instance spontaneously and in the other only after massage.

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25. Pillat, Arnold: Die Filtrationsnarben bildende Glaukomoperationen mit besonderer Berücksichtigung der Iridenkleiss antiglaukomatosa Holth, Ztschr. f. Augenh., 1928, suppl. 9, p. 1.

26. Gjessing, Harald: Ueber die Iridenkleisis antiglaukomatosa: Iridotomia meridionali nach Holth, Verhandl. d. a. Tagung d. Ophth. Gesellsch. in Wien., August, 1921, Berlin, S. Karger, 1922, p. 76.

27. Gjessing, quoted by Peters: Das Glaukom.

CHRONIC RETROBULBAR NEURITIS *

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A centenary celebration, whether of persons or of events, always creates in our young country a special interest. The privilege, therefore, of taking even a small part in celebrating the hundredth birthday of the Medical Society of Tennessee is very great, a privilege which I appreciate highly.

The subject of my address was assigned to me by your agreeable and efficient chairman; otherwise, I would never have had the temerity to attack this problem because of its voluminous literature, the confusion of chronic and acute conditions, the insertion of many cases of general optic neuritis in the reports and the variance of opinion concerning its etiology, its pathology and the best method of dealing with the lesion.

The acute form of this malady will not be included in this discussion, because it is often unilateral and rapid in development, recovery is the rule (often spontaneously), and it does not present the same problems in regard to etiology, prognosis and treatment. Consideration will be confined to those cases in which there is an involvement of the papillomacular fibers, chiefly or entirely, in contradistinction to general neuritis or perineuritis.

I shall deal with a tissue that may be considered a prolongation of the white matter of the brain. In its medullated fibers, there are no sheaths or cells of Schwann, but there are true glial cells. Hence, its reaction in disease will be like that of the central nervous system and not like that of the peripheral nerves. Cajal's [1] graphic description of the nerve cell or neuron is especially true of the optic nerve. He said: "The nerve cell or neurone is a structure which is exquisitely sensitive to normal and pathological stimuli. It is susceptible to many toxins and poisons that do not affect, or only slightly alter, the other histological elements."

This great susceptibility of the optic nerve to pathogenic agents may account for the vast number of casual factors marshaled for the explana-

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* From the Wilmer Ophthalmological Institute, Johns Hopkins University and Hospital.

* Read by invitation before the Eye, Ear, Nose and Throat Section of the Tennessee State Medical Society, at the One Hundredth Celebration of its Founding, April 8, 1930.

1. Ramón y Cajal, S.: Degeneration and Regeneration of the Nervous System, London, Oxford University Press, 1928, vol. 1.

tion of retrobulbar neuritis. They are so numerous that the wonder is that axial neuritis is not more universal. These causes will be mentioned later in the discussion of the relationship of axial neuritis to disseminated sclerosis, sinus diseases, etc.

A few of the cases that have come under my personal observation during the last few years will be reported. Owing to the difficulty of determining the existence of disseminated sclerosis in these cases, a careful survey has been made in each instance; but to avoid repetition I will enumerate here the parts examined or the types of examinations that have been made, and in each case I shall note only the positive observations: nose and throat, teeth and sinuses, roentgen examination of the chest, gallbladder, gastro-intestinal tract, skull, sella turcica and

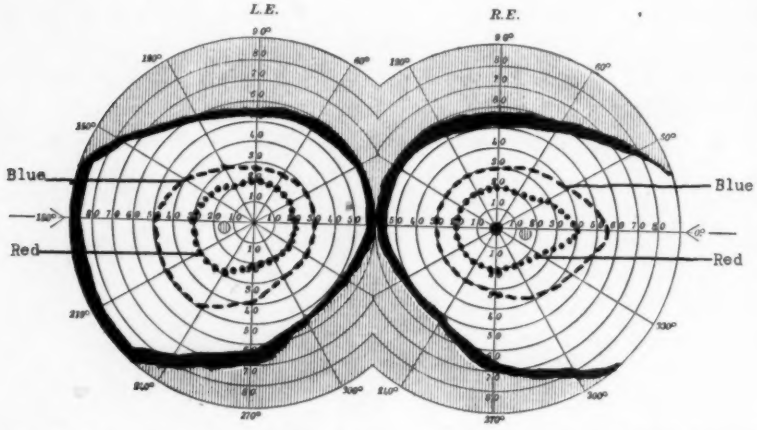


Fig. 1 (case 1).—Visual fields taken on Nov. 5, 1929. Multiple foci were present. Vision in the right eye was 20/40; in the left eye, 20/15.

optic foramen; tests of the basal metabolism, blood, blood pressure, urine, kidney function, stool, tuberculin test and spinal fluid; medical, gynecological or genito-urinary and neurologic examination.

Case 1.—*Clinical History.*—Miss E. B., white, aged 44, was admitted to the hospital for study on Nov. 4, 1929. The family history was unimportant. Her general health was good. In January, 1929, the patient noted that the sight of the right eye was becoming dim. The condition was diagnosed as optic neuritis. Two abscessed teeth were removed.

Examination.—Examination of the eyes showed them to be normal externally. Vision in the right eye with glasses was 20/40+2; in the left eye with glasses, 20/15. The right visual field showed a small relative scotoma for white and colors (fig. 1). Examination with the ophthalmoscope showed a posterior cortical cataract of the right eye; the fundus was not clearly visible. In the left eye, the media and fundus were normal.

Summary.—The patient had (1) two infected teeth; (2) sinus disease, and polypi and pus in the left ethmoid, and (3) the reaction to the tuberculin test (intradermal) was positive to 0.001 of old tuberculin.

Diagnosis.—The diagnosis was incipient cataract and retrobulbar neuritis.

Course.—The patient was seen in consultation, and the later course of the disease is not known.

Impression.—There were three possible causes of the condition, infection of the teeth and sinuses being the most likely.

CASE 2.—*Clinical History.*—C. D. C., a white man, aged 31, was admitted to the hospital for study on Feb. 27, 1928, complaining of failing vision. The family history was negative for ocular disease. The patient had been treated for syphilis, although he said that he had never had a genital lesion. The Wassermann reac-

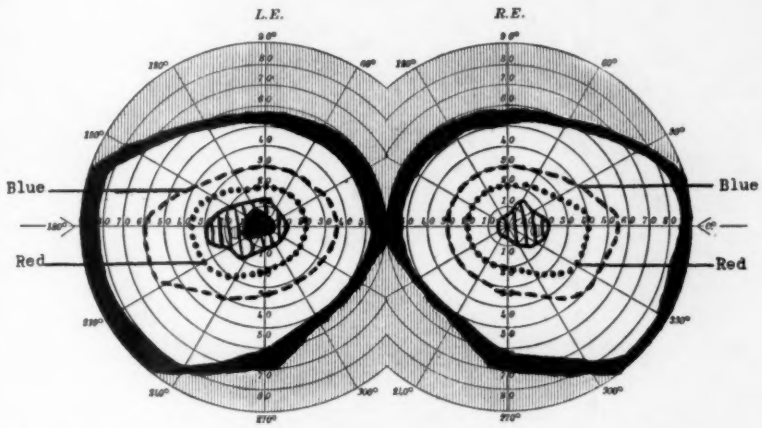


Fig. 2 (case 2).—Visual fields taken on Feb. 28, 1928. The patient had tuberculosis and cholelithiasis. Vision in the right eye was 20/200; in the left eye, 10/200.

tions of the blood and spinal fluid had been negative. In July, 1927, he accidentally noticed that the central vision in the left eye was very poor. In September, vision in the right eye began to blur, and by October was very poor. Since that time, there had been no progression. There was no definite history of the excessive use of tobacco and alcohol.

Examination.—Examination of the eyes showed them to be normal externally. Vision in the right eye was 10/100—1; in the left eye, 10/200. The visual fields were normal for form, but there was a central relative scotoma in the right eye and an absolute central scotoma in the left eye (fig. 2). Examination with the ophthalmoscope showed the media clear. There was pallor of the temporal side of the disks in both eyes, more marked in the right. Light sense was diminished (threshold).

Summary.—The reaction to the tuberculin test was positive to 0.001 mg. There were stones in the gallbladder (cholelithiasis).

Diagnosis.—The diagnosis was bilateral retrobulbar neuritis.

Course.—The patient was not seen after discharge from the hospital. Operation on the gallbladder was advised.

Impression.—In this case only two causes for the lesion of the eye seem possible: disease of the gallbladder and tuberculosis.

CASE 3.—*Clinical History.*—Mrs. M. F., white, aged 26, was admitted to the hospital on April 10, 1927, complaining of loss of vision, first in the right eye, later in the left eye. The family history was unimportant. The menstrual periods had been irregular. During the last six years, there had been a gain of 100 pounds (45.4 Kg.). Three months before examination, pain in the head and in the right eye had developed, with almost complete loss of vision for three weeks. One week prior to examination, there was a little pain in the left side of the head followed by a blurring of vision in the left eye.

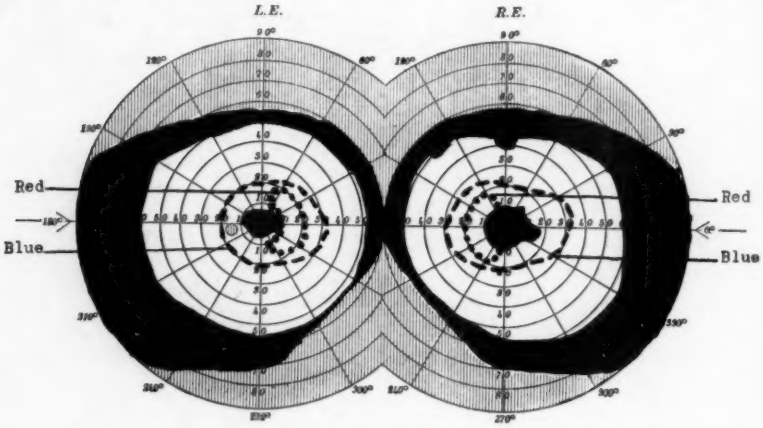


Fig. 3 (case 3).—Visual fields taken on April 13, 1927.

Examination.—Vision in the right eye was 1/200; in the left eye, 20/100. The visual fields showed slight contraction; there was an absolute central scotoma in each eye (fig. 3).

Examination with the ophthalmoscope showed: the margins of the right disk sharply defined; the nerve head white, especially on the temporal side. The disk of the left eye was normal in color; there was a slight blurring of the margins, with a slight edema in the surrounding retina.

All of the tests gave negative results. The appearance of the patient suggested endocrine disturbance (fig. 4). A gynecologist suggested the possibility of a tumor of the suprarenal glands. A neurologist suggested the possibility of a polyglandular disturbance.

Operation.—In view of the history of the right eye and the desperate condition of the vision, the ethmoids and sphenoids of both sides were opened. Cultures showed only a light growth of *Staphylococcus albus.* After this operation, there was immediate and lasting improvement in the scotoma of both eyes (fig. 5). On May 10, 1927, vision in the right eye was 20/100+1; in the left, 20/30—1. On June 9, vision in the right eye was 20/100+1; in the left, 20/15. Now (three years later), vision in the right eye is 20/70; in the left, 20/15, and there is a

central scotoma for red in the right eye. The left eye is normal. The papilla of the right eye is still pale; the fundus of the left eye is normal.

This case is interesting from the strong suggestion of pituitary disturbance and the absence of all positive observations except of the sinuses, which were only slightly infected; also because it illustrates the value of perimetry.

Impression.—The case was one of bilateral retrobulbar neuritis, probably due to sinus infection.

Fig. 4 (case 3).—Patient, showing suggestion of endocrine disturbance.

CASE 4.—*Clinical History.*—J. D. H., a white man, aged 58, was admitted to the hospital on Dec. 9, 1925, complaining of loss of sight. The family history revealed that one aunt and one uncle had died of tuberculosis. No eye trouble was reported. Seven years before examination, there was a discharge from the ears; the tonsils were removed in 1924. Vision had become very much blurred; there was a slight hemorrhage in the right eye. Eleven bad teeth had been removed; ten days later, slight hemorrhage occurred in the left eye.

Examination.—Examination of the eyes showed them to be normal externally. Vision in the right eye was 2/200; in the left, 10/200. The visual field of the right eye presented a large central scotoma; that of the left eye, a small central scotoma (fig. 6). Examination with the ophthalmoscope revealed: media, clear;

the temporal side of the disk, white; the vessels, of normal size. There were minute vitreous opacities in the left eye; a small hemorrhage above, and to the outer side of, the disk, and a slight paleness of the temporal side of the disk.

Summary.—In addition to the infected (streptococcus) teeth removed, there was some pus and a cheesy polypoid mass in the left ethmoid; the roots of the

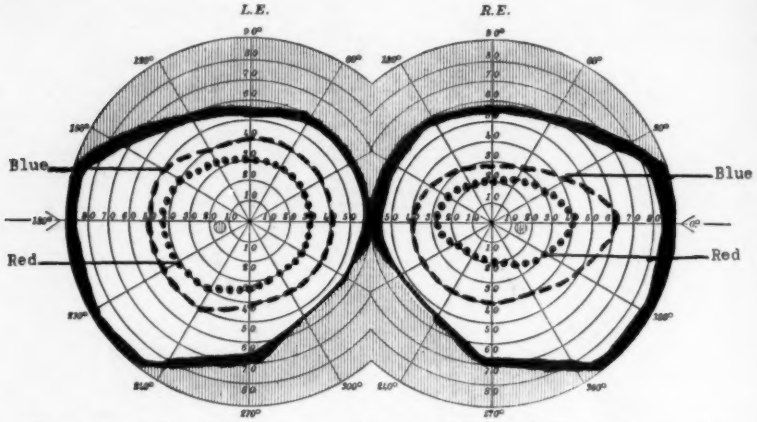


Fig. 5 (case 3).—Visual fields taken on May 10, 1927, after operation.

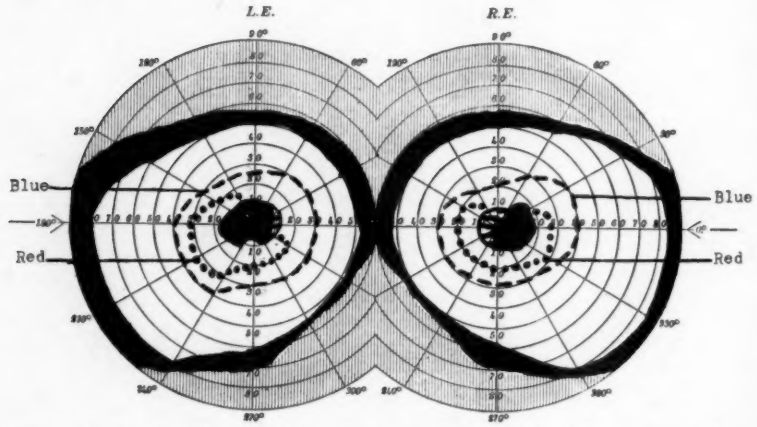


Fig. 6 (case 4).—Visual fields taken on Jan. 17, 1930, four and one-half years after observation. Multiple foci were present. Vision in the right eye was 8/200; in the left eye, 10/200.

removed tonsils were badly infected. In the left one there was much pus, a culture of which showed beta hemolytic streptococcus. Later, a fibroid appendix was removed; no pus was obtained.

At the time of writing of this article, vision in the right eye was 8/200; in the left, 10/200. The scotoma remained in both eyes.

Impression.—The cause of the axial neuritis in this case must be attributed to the teeth, tonsils and sinuses.

CASE 5 (Courtesy of Dr. Cecil H. Bagley).—*Clinical History.*—Miss R. M., white, aged 19, was admitted to the hospital on Aug. 24, 1927, complaining of poor vision. The family history was unimportant. At times there had been difficulty in controlling the left foot. One year before admission, the patient noted difficulty in reading. In June, 1927, vision in the left eye became impaired. This was followed a few days later by a similar condition in the right eye. The condition was diagnosed as retrobulbar neuritis. Infected tonsils were removed, and the sphenoids opened; this operation was followed by slight improvement in vision. Vision in the right eye was 6/200; in the left, 3/200.

Examination.—Examination of the visual field showed an absolute central scotoma in each eye (fig. 7). Examination with the ophthalmoscope revealed:

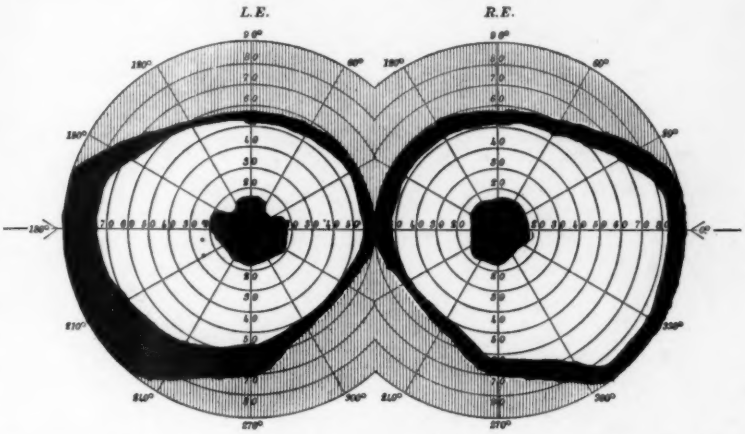


Fig. 7 (case 5).—Visual fields taken on Aug. 24, 1927. Vision in the right eye was 6/200; in the left eye, 3/200.

the fundus of each eye normal, except for slight pallor of the temporal side of the disks.

Summary.—All of the tests gave negative results, except for a low grade infection of the sinuses already operated on.

On Aug. 24, 1929, vision in the right eye was 20/70; in the left, 20/70. The central scotoma were merely relative (fig. 8). The color of the disk was improved. A careful neurologic examination failed to reveal any evidence of disseminated sclerosis.

Impression.—Disseminated sclerosis may develop later, but up to the time of writing this article the sinus infection seemed to be the chief factor in the cause of the retrobulbar neuritis.

CASE 6.—*Clinical History.*—J. N., a white man, aged 22, was admitted to the hospital on June 5, 1929, complaining that his vision had been impaired for a month previously. A brother two years older than the patient had suffered from

impaired vision for ten years due to some optic nerve trouble. (Since then an examination has shown that he is suffering from retrobulbar neuritis [fig. 9]). One month before admission, the vision in the left eye became dim. This impairment progressed so rapidly that within two weeks the patient could not see light straight ahead. Two weeks before examination, the right eye became involved in the same manner. He could see objects on the side but nothing straight ahead. Vision in the right eye was 8/200; in the left eye, 2/200.

Examination.—The visual fields showed a large central scotoma in each eye (fig. 10). Examination with the ophthalmoscope revealed: media, clear; the nerve heads, normal in color and outline; a marked physiologic cup. In the right eye, there was a small hemorrhage below and to the nasal side of the macular region.

At the time of writing this article, the temporal side of each nerve was white. While the sight had not improved, the patient had adjusted himself to the loss of central vision and got along very well.

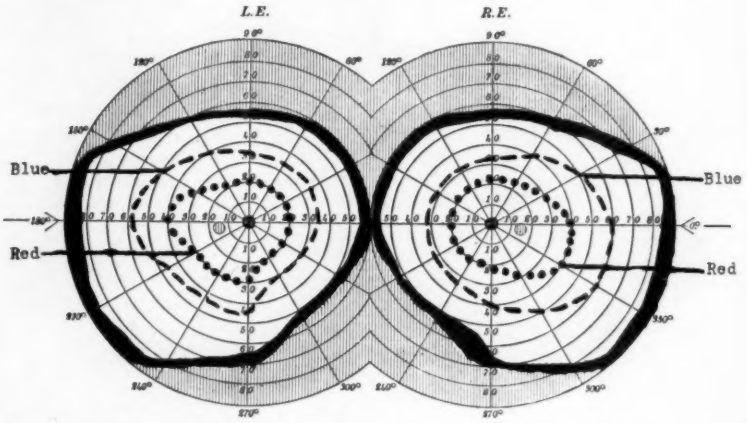


Fig. 8 (case 5).—Visual fields taken on Aug. 24, 1929. Vision in the right eye was 20/70; in the left eye, 20/70.

Summary.—The ethmoid contained polypi; there was no pus and no growth from cultures. There was infection of the tonsils and adenoids.

Impression.—The family history, with the observations of a similar condition in the eyes of an elder brother, leaves little doubt but that this case belongs to the group of familial optic atrophy—Leber's disease (fig. 11).

Through the courtesy of Dr. John M. Wheeler, I have just seen a young man, aged 28, whose eyes present the same picture of Leber's disease as appears in the two brothers just mentioned (fig. 12).

This patient gave the following family history: One uncle had the same eye condition as the patient. A maternal uncle, who died at the age of 34, and a first cousin of the patient's mother also had eye trouble.

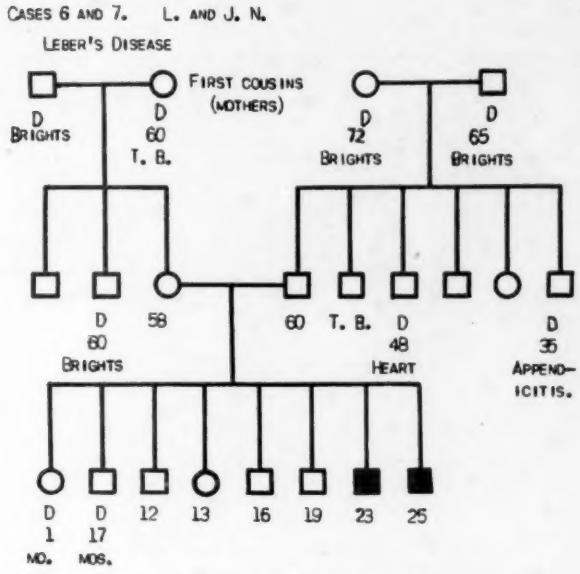


Fig. 9.—Chart showing familial diseases.

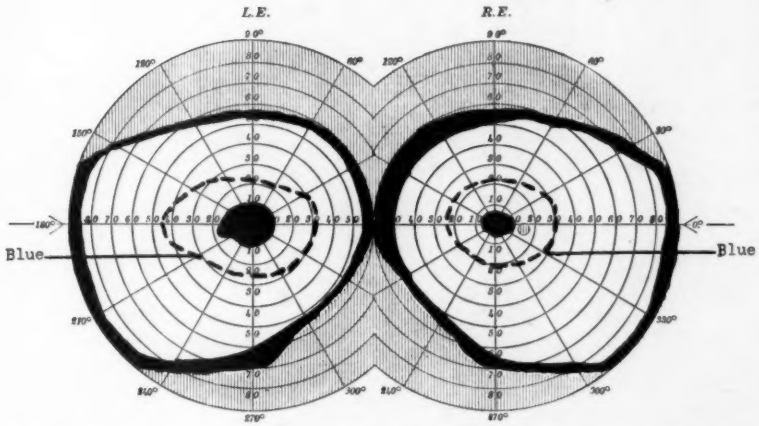


Fig. 10 (case 6).—Visual fields taken on June 6, 1929. The patient had Leber's disease. Vision in the right eye was 8/200; in the left eye, 2/200.

CASE 7.—*Clinical History.*—H. S., a white man, aged 58, was first seen on Dec. 14, 1926, when he complained of blurring of vision, especially in the right eye. The family history was irrelevant. There had been an abscess of the rectum, which occurred about the same time as the impairment of vision. In November, he consulted an oculist on account of changing blurred spots in the

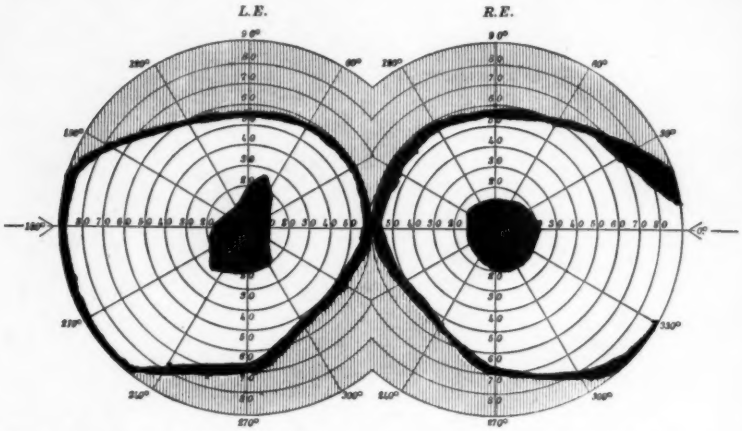


Fig. 11 (case 6).—Visual fields taken on March 17, 1930. Vision in the right eye was ½/200; in the left eye, 1/200.

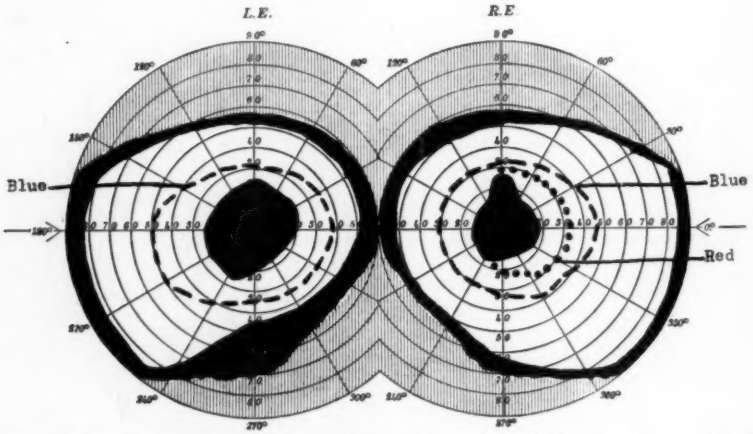


Fig. 12 (case 6).—Visual fields taken on Nov. 25, 1929. Vision in the right eye was 4/200; in the left eye, 2/200.

field of the right eye. The vision decreased in a few days from 20/20 to 20/30. Both antrums were opened, and the vision soon became normal, but did not remain so. The patient smoked three pipes and two cigars a day, and he used alcohol moderately. Vision in the right eye was 20/30+2; in the left eye, 20/20. Light sense was reduced in both eyes, but more so in the right.

Examination.—The visual fields showed a relative scotoma for form and an absolute scotoma for color in each eye (fig. 13). Examination with the ophthalmoscope revealed: the upper nasal margins of the right disk blurred; color, normal. The arteries were a trifle tortuous. The fundus of the left eye was normal.

Summary.—The positive observations were numerous. There was infection of the sinuses, particularly of the right antrum. The tonsils were infected (*Streptococcus aureus* and *albus*). The prostate and processes of the lumbar vertebrae showed an infectious process. The patient was sensitive to tuberculin, 0.0001 mg.

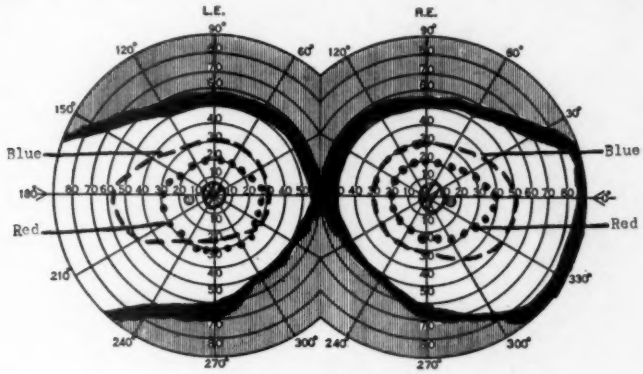


Fig. 13 (case 7).—Visual fields taken on Jan. 11, 1926. Vision in the right eye was 20/30+2; in the left eye, 20/20.

On Jan. 8, 1928, vision in the right eye was 20/15—1; in the left eye, 20/15; only a small, paracentral scotoma for red remained in each eye (figs. 14 and 15).

Impression.—With the multitude of possible sources of infection, it is impossible to say which is the cause of the lesion of the eye. In spite of the removal of the infected foci, the vision improved only slightly;

but a decided and lasting improvement commenced after the use of tuberculin therapeutically.

It seems at least probable that a tuberculous toxemia may have exerted some influence on the condition.

CASE 8.—*Clinical History.*—Miss M. A. C., white, aged 20, was admitted to the hospital on July 30, 1929, complaining of blurring of vision in the left eye

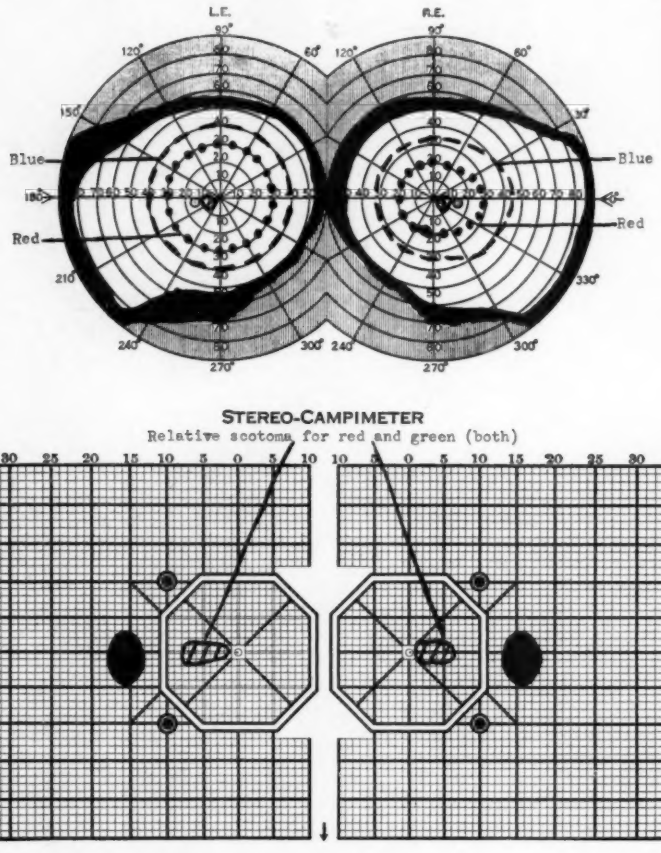


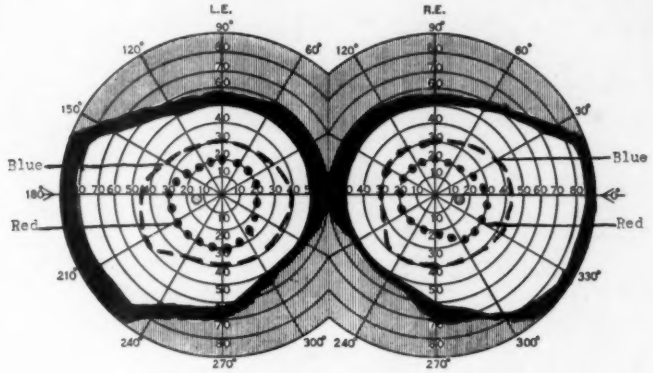
Fig. 14 (case 7).—Visual fields taken on March 30, 1927. Vision in the right eye was 20/20; in the left eye, 20/20+1.

for the past week. There was a history of alcoholism in the father. Appendectomy had been performed in December, 1926. There was only occasional sore throat. She had had one attack of tonsillitis two years before examination. There had been a slight swelling of the ankles for the past three months.

One year prior to examination, she had blurring of vision in both eyes for several days. This disappeared completely after she had worn glasses regularly. Nine days before examination, pain occurred over the left eyeball. This became

noticeable on pressure over the eye and on motion. One year previous to examination, objects began to be blurred, and these symptoms had since become worse. There was no disturbance in the right eye. There had been no headache. She was taken to the hospital with a temperature of 101 F., but it fell shortly after admission and remained normal. There was slight nausea, but no vomiting. Vision in the right eye was 20/20; in the left eye, 20/40.

Examination.—The visual fields of the right eye were normal for form and color. There was a concentric contraction in the field of the left eye, particularly



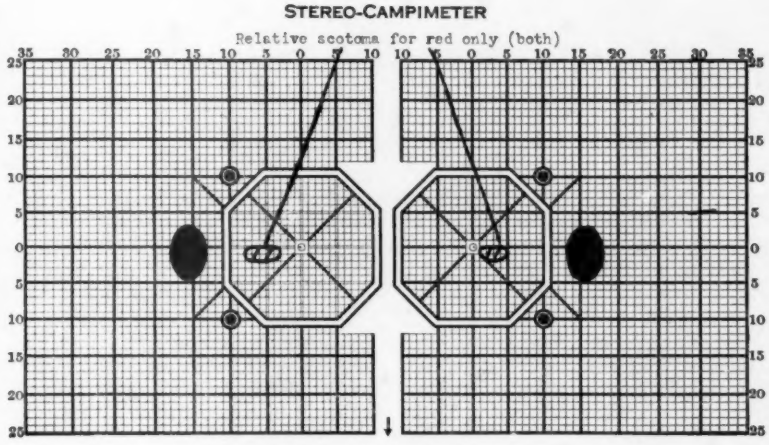


Fig. 15 (case 7).—Visual fields taken on June 8, 1928. Vision in the right eye was 20/15—1; in the left eye, 20/15.

of the upper temporal quadrant. There was a central scotoma (relative) for red (fig. 16). Examination with the ophthalmoscope revealed the right eye to be normal. In the left eye, 3 disk diameters below and slightly to the temporal side of the disk, there was a large coloboma of the choroid and retina. On the temporal margin of the disk, there was a small colloid excrescence. The papilla itself was slightly hyperemic, and the margins were a trifle blurred.

Six months later, the vision, fields and other organs were practically unchanged, but the scotoma was less (fig. 17).

Summary.—The tuberculin reaction was positive to 1.001 mg. The tendon reflexes were more active on the left than on the right side. The spinal fluid showed a positive globulin with a negative Wassermann reaction.

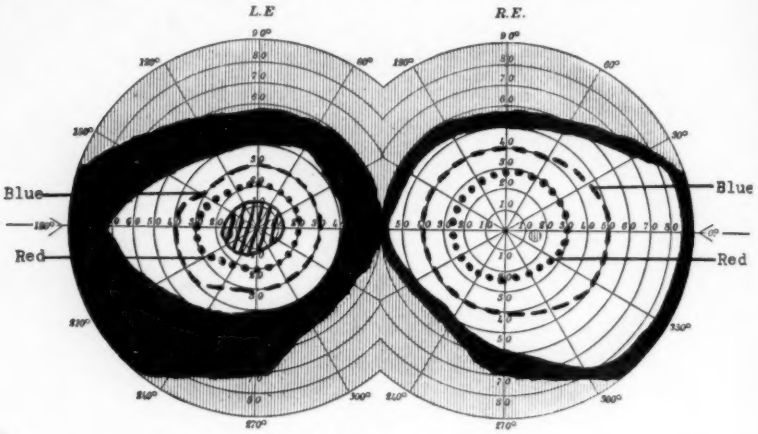


Fig. 16 (case 8).—Visual fields taken on July 30, 1929. The patient had disseminated sclerosis. Vision in the right eye was 20/20; in the left eye, 20/40.

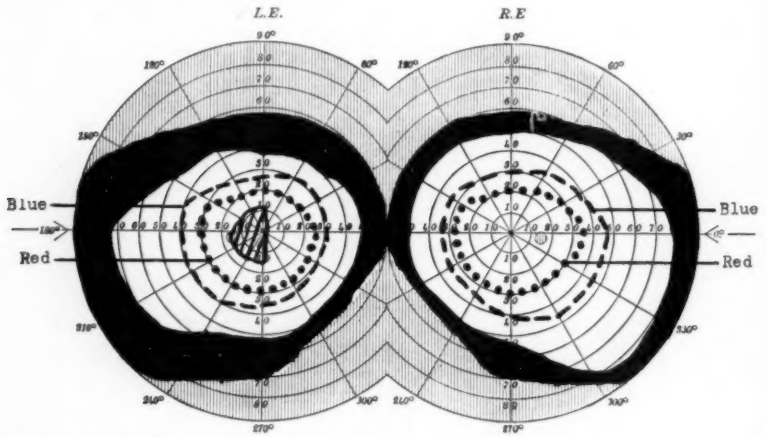


Fig. 17 (case 8).—Visual fields taken on Jan. 12, 1930. Vision in the right eye was 20/20; in the left eye, 20/30.

Impression.—The case was unilateral axial neuritis with a probable beginning disseminated sclerosis.

COMMENT

Symptoms.—In the earlier stages of axial neuritis, the patient complains of a cloud or a blur in the center, or near the center, of vision.

Later this becomes a definite blind spot. The patient sees better in a moderate light than in a very bright one. The vision improves after a period of rest, and it is temporarily helped by inhalations of amyl nitrite. Later, the patient develops a habit of tilting his head, or of using his eyes eccentrically to see around the obstruction. According to Percival,[2] the light sense difference is much impaired, while the light sense minimum (threshold) is little impaired. In my experience, both are reduced. The visual acuity is at times reduced as low as 1/200. In disseminated sclerosis, there is great variability in the scotoma. The perimeter, or campimeter, gives the most accurate information of the extent of the involvement of the papillomacular fibers. In the early stages, the scotoma is relative and affects particularly the perception of green and red. Later blue and white are not recognized, and the scotoma becomes absolute. There may also be irregular contractions of the peripheral field. Associated perineuritis may add peripheral field defects to the central changes. According to Traquair,[3] "The central defects in sinus disease are not characteristic, but their study is of great value from a negative standpoint in enabling one to distinguish them from tobacco amblyopia or defects from pituitary tumor."

In this connection Cushing's [4] remarks on the "chiasmal syndrome" are illuminating. He said: "This condition is of particular interest to the ophthalmologist, for he will naturally be the first person consulted for failing vision in the absence of other symptoms, and the diagnosis of toxic amblyopia (retrobulbar neuritis, etc.) is likely to be made."

In Leber's disease, the central field defect is usually round or oblong, occasionally irregular; it is absolute and permanent. According to Hensen,[5] "the central scotoma in disseminated sclerosis is very fleeting; in fact, disseminated sclerosis never causes a scotoma of longer than three months' duration." This changing vision is well illustrated by the following letter, which I received recently from a woman concerning her son: "Last July he started to lose the sight of one eye. About two months later the other eye started to grow dim. He has had great trouble in seeing ever since. The sight comes and goes, at one time being much better than at another. He has been to see eye specialists

2. Percival, A. S.: Retrobulbar Neuritis and Associated Conditions, Tr. Ophth. Soc. U. Kingdom **46**:392, 1926.

3. Traquair, H. M.: Clinical Perimetry, London, Henry Kimpton, 1927, p. 115.

4. Cushing, Harvey: The Chiasmal Syndrome: Of Primary Optic Atrophy and Bitemporal Field Defects in Adults, with a Normal Sella Turcica, Arch. Ophth. **3**:505 (May) 1930.

5. Hensen, H.: The Importance of the Duration of the Central Scotoma in Cases of Retrobulbar Neuritis for the Diagnosis of Multiple Sclerosis, Klin. Monatsbl. f. Augenh. **72**:75, 1924.

and physicians. They could not find out the cause of the trouble. About three weeks ago he began to have trouble with his legs. He could not balance himself and would fall down anywhere. He went to a neurologist, who told us that our son had multiple sclerosis." This description was given seven months after the onset of the visual symptoms.

Fundus.—At first, as a rule, the fundus appears normal, or there may be a slight blurring of the margin of the disk with a definite hyperemia and fullness of the veins. Later, the temporal side of the nerve becomes pale, or the whole nerve appears atrophic in spite of a good peripheral visual field. The preexisting physiologic cup is much increased by the atrophy of the nerve fibers. Several times in severe cases with much reduced vision, I have noted small hemorrhages in the retina approximately between the papilla and the macula. Actual swelling of the disk has been noted by some observers.

In cases of tumors of the frontal lobe, both Kennedy [6] and Lillie [7] have noted the occurrence of retrobulbar neuritis on the side of the tumor and a true papilledema in the other eye. Time will not permit the discussion of these interesting conditions.

Pathologic Changes.—The following changes may be recognized in the orbital portion of the optic nerve: atrophy of the axis cylinders, inflammatory changes in the trabeculae of the connective tissue, thickening of these fibers, sclerosis of the walls of the small nutrient blood vessels and proliferation of the glial cells. Some observers (Marburg [8]) believe that the axis cylinders and the ganglion cells of the retina are involved primarily, while others hold the view that the changes in the nervous elements are secondary to a preexisting inflammation. In the case of the so-called toxic amblyopias, it is generally thought that the primary change is in the ganglion cells of the macular region of the retina, and that the papillomacular bundle is secondarily affected (ascending retrobulbar neuritis). Observers of experience are of the opinion that the axial fibers of the optic nerve are particularly highly organized and therefore especially vulnerable to endogenous toxins, ectogenous poisons, pressure and inflammatory processes. Traquair [3] expressed the opinion that the changes in the optic nerve may result from "venous congestion of the nerve, leading to impaired nutrition and suffocation of the nerve fibers." Percival [2] called attention to the obser-

6. Kennedy, F.: Retrobulbar Neuritis as an Exact Diagnostic Sign of Certain Tumors and Abscesses in the Frontal Lobes, Am. J. M. Sc. **142**:355, 1911.

7. Lillie, Walter I.: Ocular Phenomena Produced by Basal Lesions of the Frontal Lobe, J. A. M. A. **89**:2099 (Dec. 17) 1927.

8. Marburg, O.: Retrobulbar Neuritis and Multiple Sclerosis, Ztschr. f. Augenh. **44**:125, 1920.

vation of Samelsohn of the part played by the optic foramen in constricting the optic nerve. Herzog [9] expressed the opinion that the cases of retrobulbar neuritis of nasal origin are really circumscribed serous meningitis which spreads to the optic nerve from the diseased sinuses through narrow communications.

In Leber's disease, the condition may be a true abiotrophy similar to the changes in the retina in so-called "retinitis pigmentosa." I do not know of any instance in which there has been a microscopic examination of the optic nerve in this disease.

Disseminated Sclerosis.—Price [10] said: "Apart from syphilis, disseminated sclerosis is the commonest organic disease of the nervous system in English and Continental practice. It is said to be less frequent in America." Indeed, the causes of retrobulbar neuritis and disseminated sclerosis, as given by various authors, are interchangeable, as will be seen from the following parallel columns:

Causes of Disseminated Sclerosis	Causes of Retrobulbar Neuritis
Acute infectious diseases (typhoid, smallpox, scarlatina, measles, etc.), influenza, cholera, whooping cough, acute articular rheumatism, puerperium, malaria, diphtheria, lightning, intoxication with metallic poisons, tin, carbon monoxide, mercury, alcoholism, injury, violent emotion, overstrain, chill, shock, toxins (disputed by some observers), endogenous and constitutional (Oppenheim)	Acute infectious diseases (typhoid, smallpox, scarlatina, measles), influenza, cholera, whooping cough, acute articular rheumatism, puerperium, malaria, diphtheria, various chemical poisons, injury, emotional strain, chill, infection of the tonsils and teeth, sinus disease, gastro-intestinal intoxication, anomalies in development, *Bacillus subtilis* (Clark), acetonuria (Francis), pellagra (Calhoun), diabetes, albuminuria, tuberculosis (?), embolism (various authors)

Traquair [3] considered disseminated sclerosis as the cause of retrobulbar neuritis in from 20 to 40 per cent of the cases; Marburg,[8] in from 50 to 55 per cent; Scheerer,[11] as actual or probable, in 71.5 per cent, and Langenbeck [12] in 77 per cent. In this country there seem to be fewer cases of disseminated sclerosis associated with retrobulbar neuritis. Indeed, in some localities disseminated sclerosis itself seems less fre-

9. Herzog, H.: Retrobulbar Neuritis, Arch. f. Augenh. **292**:321 (July) 1928.

10. Price, F. W.: A Textbook of the Practice of Medicine, ed. 3, London, Oxford University Press, 1929.

11. Scheerer, R.: Causation of Retrobulbar Neuritis, Klin. Monatsbl. f. Augenh. **83**:164 (Aug.-Sept.) 1929.

12. Langenbeck, quoted by Cords, F. C.: Critical Observations on Rhinogenic Retrobulbar Neuritis, Klin. Monatsbl. f. Augenh. **69**:129, 1922.

quent. Of the 12,000 autopsies made in the department of pathology at Johns Hopkins University Medical School, there were only two cases of disseminated sclerosis. Of course, one must consider that few patients with disseminated sclerosis die in hospitals.

Of the patients in the Institute for study, 0.59 per cent, or thirty cases, had been affected with retrobulbar neuritis. I have followed closely the history of eighteen of the thirty cases. One of them is associated with disseminated sclerosis and one suggests that possibility.

The island-like lesions in the brain, spinal cord and cranial nerves appear as grayish-blue plaques, varying from a fraction of a millimeter to 10 mm. in diameter. The white matter is chiefly involved, and, according to Price,[10] the inflammatory changes are the initial lesions, and the alterations in neuroglia and nerves are secondary. There are edema, cellular infiltration, increase of interstitial connective tissue, thrombosis of vessels with formation of new ones and disintegration of the myelin sheath of the nerve cylinders. The essential feature of the process is the great persistence of the axis cylinders with absence of secondary degeneration, which accounts for the many and varying symptoms. According to Zeigler,[13] there is a congenital tendency to proliferation of glial tissue in these cases.

Sinus Infection.—The relation of diseases of the paranasal sinuses to axial neuritis is especially confused. Its estimation as a cause of axial neuritis varies all the way from being considered negligible to being held as the chief factor.

Onodi [14] stated that more complete study resulted in the establishment and confirmation of thirty-eight forms of morphologic relations of the optic nerve to the sphenoid sinuses and the posterior ethmoid cells.

Sinus disease is common in this country. Perhaps this is due to the great difference between the dry air of the overheated houses and the cold, humid outside air of winter, for it requires a rapid adjustment on the part of the nasopharyngeal membrane to meet the changes from a house temperature of 70 F. with a humidity of 30 degrees to an outside atmosphere of 20 F. with a humidity of 87 degrees.

Is it not possible that there may be a causal relation between diseased sinuses and disseminated sclerosis, the infectious agent gaining entrance to the central nervous system through the sinuses? The fact that so far no pathogenic agent has been isolated in disseminated sclerosis suggests the possibility that this agent may belong to the group of so-called

13. Zeigler, quoted by Oppenheim, H.: Text Book of Nervous Diseases, ed. 5, New York, G. E. Stechert & Company, 1911, vol. 1, p. 332.

14. Onodi, A.: The Optic Nerve and the Accessory Cavities of the Nose, Ann. Otol. Rhin. & Laryng. **17**:1 (March) 1908.

"filtrable viruses." The prompt and radical sinus operative procedure in this country may account for the fact that disseminated sclerosis is less frequent here than abroad. Markbreiter, as quoted by Herzog,[9] found sinus disease to be the cause of 79 per cent of the cases of axial neuritis, while Edwin Wallis (also quoted by Herzog) found the percentage as high as 90 per cent. In this country, Oliver and Crowe[15] stated that "infection of the accessory nasal sinuses is one of the common causes of retrobulbar neuritis." But they added this significant sentence: "On the other hand, only a small percentage of patients with infection of the sinuses have a retrobulbar neuritis as a complication."

In contradistinction to the feeling that so many cases of axial neuritis are due to sinus disease, Weill[16] noted the fact that 85 per cent of his patients recovered without surgical interference, which he thinks precludes the possibility of their being caused by the sinuses. In England, the general opinion was expressed by Mayou,[17] who said: "If it (sinus trouble) was a cause of retrobulbar neuritis, it was a rare one, with which six of my colleagues agree."

Leber's Disease.—In Leber's disease also, the patients are generally youthful. In this condition, as well as in disseminated sclerosis, there may be mental and nervous diseases. But it is helpful in making a diagnosis to realize that the large majority of those affected are males, the onset of the disease is rapid, the scotoma is permanent and, as a rule, peripheral field defects, so often seen in disseminated sclerosis, are absent. The family history of other male members of the family with similar eye trouble is also helpful in making a diagnosis. As in retinal abiotrophy, the onset of the disease is influenced by certain outside factors, such as serious illnesses, focal infections and toxemias.

Teeth, tonsils and other foci of infection often play a part in axial neuritis. In forty-six cases White[18] found the cause of the disturbance of the optic nerve to be due to the teeth in 30 per cent, the tonsils in 33 per cent and a combination of teeth and tonsils in 10 per cent. In my own experience, infected teeth and tonsils often occur with other possible causes.

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15. Oliver, K. S., and Crowe, S. J.: Retrobulbar Neuritis and Infection of the Accessory Nasal Sinuses, Arch. Otolaryng. **6**:503 (Dec.) 1927.

16. Weill, Georges A.: The Relationship Between Inflammation of the Posterior Sinuses and Disease of the Nervus Opticus, Arch. Ophth. **1**:307 (March) 1929.

17. Mayou, M. S.: Discussion of Optic Neuritis in Its Relation to Sinusitis, Proc. Roy. Soc. Med. **19**:57, 1926.

18. White, Leon E.: The Location of the Focus of Optic Nerve Disturbances from Infection, Ann. Otol. Rhin. & Laryng. **37**:128, 1928.

Of the 5,058 patients admitted to the institute for survey, 30 suffered from retrobulbar neuritis. Of these, 18 cases have been followed up for some time, and the following tabulation gives the probable cause:

Multiple sclerosis probable	11 per cent
Sinus disease probable	11 per cent
Gallbladder probable	6 per cent
Diabetes probable	6 per cent
Leber's disease	16 per cent
Tuberculosis possible	6 per cent
Multiple foci (teeth, tonsils and sinuses)	44 per cent

Fifty per cent of these patients were sensitive to tuberculin. In one case, the cause is put down as probable tuberculosis, because the vision and the scotoma did not improve after the foci of infection had been removed; improvement followed the therapeutic use of tuberculin. This improvement has been permanent.

CONCLUSIONS

1. In all cases of axial neuritis a detailed family history is helpful.

2. A complete survey should be made, including an examination of the spinal fluid in all doubtful cases. A positive curve with a negative Wassermann reaction is highly suggestive of disseminated sclerosis.

3. Frequent painstaking perimetric measurements are essential features of the study of these cases.

4. In disseminated sclerosis, axial neuritis may appear late in the disease; but, again, it may be the first symptom that brings the patient to the physician. Consequently, in cases that are classed as due to sinus disease or some other focus of infection disseminated sclerosis may develop later.

5. In both disseminated sclerosis and Leber's disease the subjects are young and nervous, and mental defects may be present. The family history, the sex and the constant absolute central scotoma in Leber's disease are helpful in making a differential diagnosis.

6. The possibility of diseases of the pituitary body must always be kept in mind.

7. In the case of many possible sources of axial neuritis, it is often impossible to name the only and essential one.

TREATMENT

1. Treatment consists in the removal of any manifest focus of infection and treatment for any discovered toxemia, such as syphilis, tuberculosis and diabetes.

2. In chronic axial neuritis in which no source of infection can be found, it is legitimate to explore the sphenoidal and posterior ethmoidal cells for the purpose of diagnosis.

3. In Leber's disease and disseminated sclerosis, frankly infected sinuses should be drained and other foci of infection removed.

4. In the present knowledge, or rather want of it, in regard to disseminated sclerosis, there is but little of specific therapeutic value that can be suggested. General hygiene and regimen are important.

5. In Leber's disease, the greatest help lies in telling the patient frankly of his condition and in helping him to face life with the prospect of impaired vision. Healthful employment is most desirable. The patient should be told that he will not be blind, and that at 50 he will probably be better able to adjust himself to the central blindness than at the present time.

OPACITIES OF THE VITREOUS*

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NEW YORK

This paper is the result of a survey of the histopathology of the vitreous body so far as it relates to the ordinary opacities seen in life.

Many fine floating opacities seen clinically are not visible in microscopic preparations. Tiny filaments are observed in life because their index of refraction is higher than that of the vitreous, in the same manner as threads of glass are outlined when suspended in water. In the preparations, however, these same opacities are rendered absolutely invisible by Canada balsam just as rods of glass are not so readily outlined in gasoline or oil. On the contrary, cells, which have a higher refractive index, are revealed under the microscope while the delicate homogenous fibers or lamellae to which they may be clinging may not be visible. The muscae volitantes of young persons, which are likened to strings of pearls, are probably cells adhering to fine threads too transparent to be seen.

CLASSIFICATION OF OPACITIES

With a view to systematic study, the opacities are divided as follows:

1. *Physiologic Opacities.*—(*a*) Embryonal: These have mostly to do with the remains of Cloquet's canal or of the hyaloid artery. They are often as difficult to differentiate microscopically as clinically from the products of inflammation or degeneration.

In one specimen, enucleated shortly after an injury, the walls of a large blood vessel which extended far out into the vitreous from the nerve head were infiltrated with lymphocytes, as were the walls of the adjacent retinal vessels. Only because of the early enucleation and because the cells were arranged in concentric rows could the diagnosis of persistent hyaloid artery be made with certainty.

(*b*) Senescent: In old age the delicate meshwork of the center of the vitreous, which is of a different texture from the stroma in the periphery, breaks down and disappears, so that areas of clear fluid result.

Frequently an elderly person will complain of a "fly" that floats before the field of vision. He states that it appeared suddenly, that it is homogeneous, that it never changes its form and that it always moves within fixed limits. Formerly these symptoms were thought to be due to minute hemorrhages because of their sudden onset. The "fly"

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* Read before the Oxford Ophthalmological Congress, July 10, 1930.

may be only a thread or a tangle of threads partly or wholly detached from the wall of a cavity. This is not alarming. It is seen with the ophthalmoscope, but it is not perceptible in the sections.

2. *Pathologic Opacities, According to Shape and Size.*—(*a*) Cells from Exudation, such as Lymphocytes and Leukocytes: Normally a few cells may always be present in the vitreous, but, as in other tissues, when they are numerous they represent disease. One is familiar with

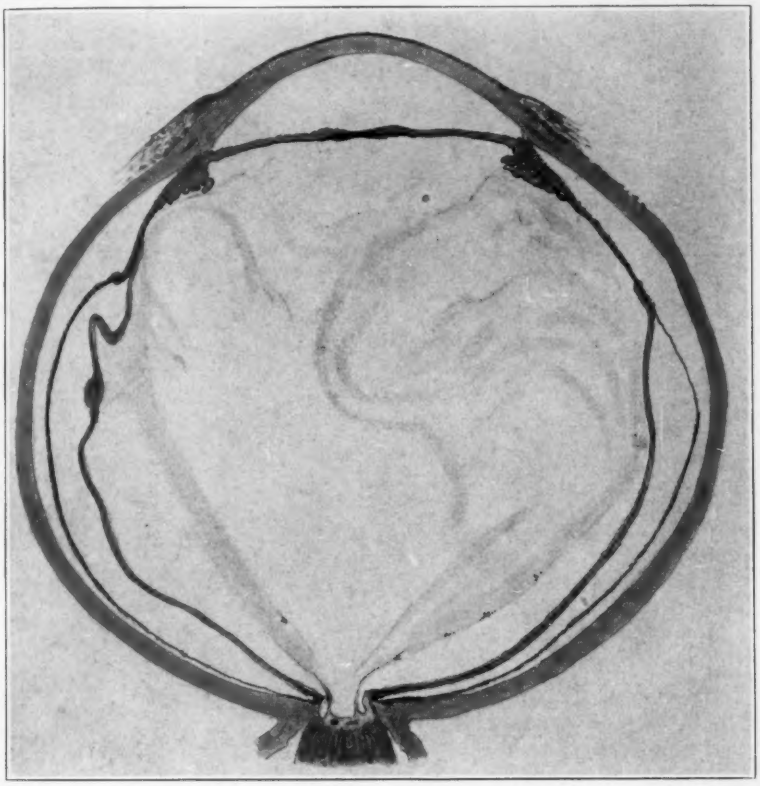

Fig. 1.—The posterior limiting layer of the vitreous is drawn into the excavated nerve head.

the phenomenon of a vitreous so cloudy one day that the patient cannot count fingers and yet in a week or two vision may be back to normal, so rapidly may cells disappear from the vitreous.

Exudates that are really in the vitreous may be erroneously located with the ophthalmoscope in the retina. The small round "woolly" opacites often referred to as being in the retina may prove under the microscope to be aggregations of lymphocytes that have broken through the limitans interna of the retina and the posterior limiting layer of the

vitreous. They are spread out in the form of mounds lying on the inner surface of the posterior limiting layer of the vitreous. They are not as sharply defined as infiltrations in the meshes of the retina, and they disappear more rapidly.

Cellular elements may be caught between the limitans interna and the posterior limiting layer and form a narrow zone of great extent.

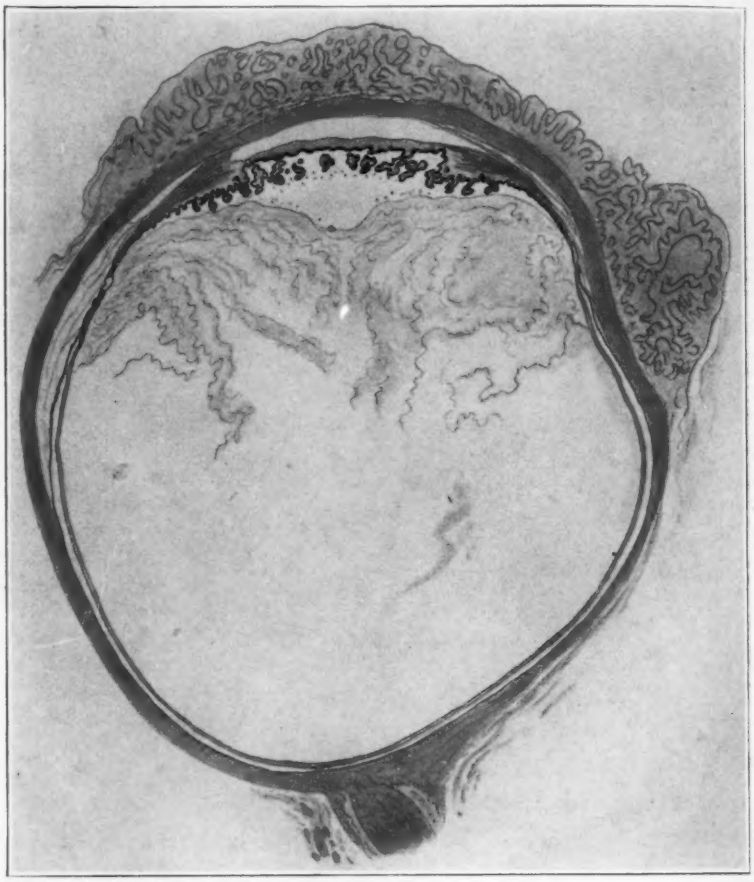

Fig. 2.—A case of high myopia. All that remains of the stroma is concentrated anteriorly in the form of wavy membranes.

Such a covering of round cells gives the retina a uniformily cloudy appearance.

In one instance after an injury followed by infection the entire vitreous body was separated from the retina by an albuminous fluid containing cells. The posterior limiting layer of the vitreous and the wall of Cloquet's canal were sharply defined. It is evident that they

formed barriers to migrating cells. The posterior limiting layer impeded the entrance of cells into the vitreous from the retina, while the cells that had gained access into the vitreous body were prevented from entrance into Cloquet's canal by its wall. The limitans interna itself

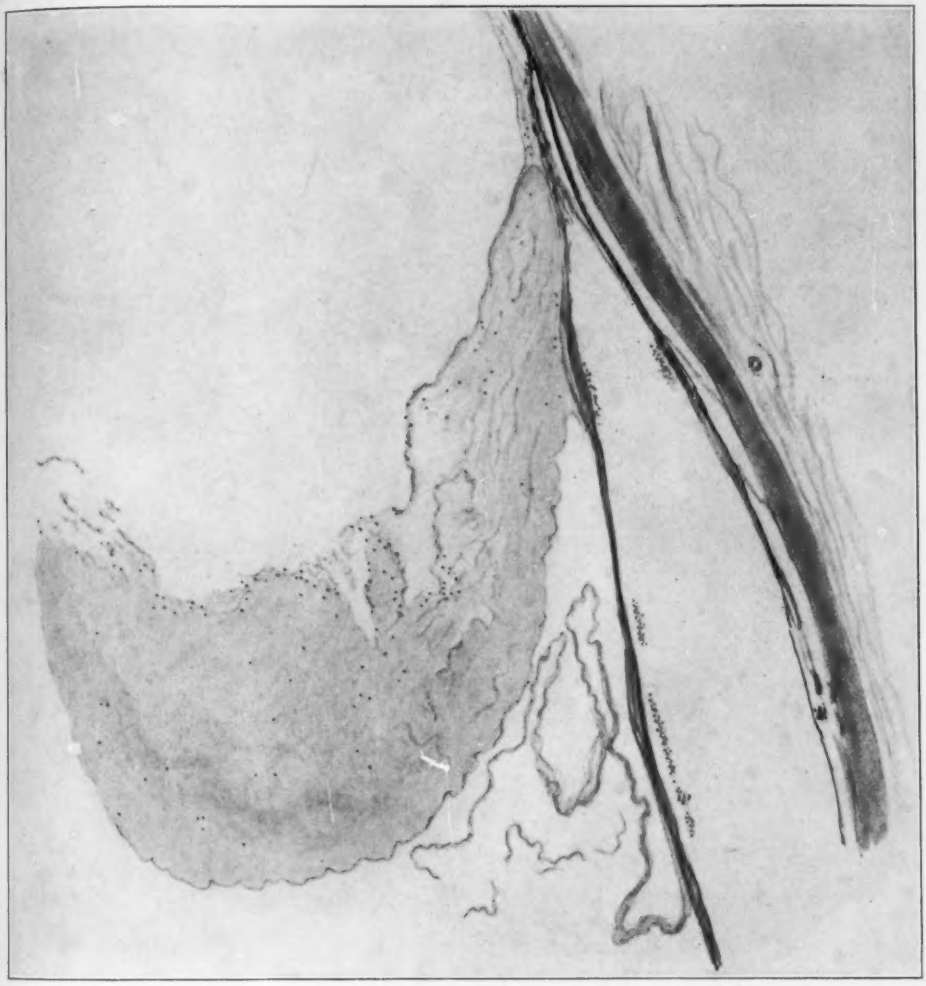

Fig. 3.—By traction such remnants of the vitreous body may cause detachment of the retina.

lay in undulations over the retina with round cells between it and the nerve fiber layer.

Cellular elements in the vitreous are regularly disposed according to the structure of the stroma. A familiar sight at the periphery is wandering cells or erythrocytes arranged in parallel lines in a sagittal

direction; the lamellae that bring about this arrangement, however, are often too delicate to be demonstrated in the sections. Similar parallel rows are observed posterior to the lens, running in a frontal direction and evidently brought about by the radiating lamellae. In one preparation a sheet of red blood cells with a few leukocytes was cut vertically across just in front of the retina, from which it had floated away. The cells appeared to be held together, not by any membrane, but by the blood plasma itself.

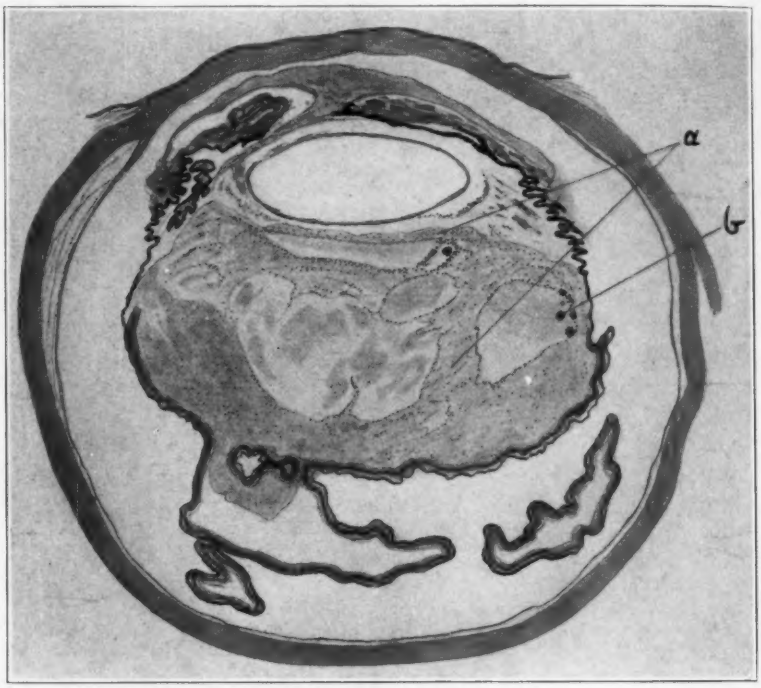

Fig. 4.—*a*, stroma diffusely infiltrated with lymphocytes; *b*, cavity showing precipitates on its wall.

In a specimen from the eye of a man, aged 69, enucleated because of spontaneous iritis with glaucoma, characteristic senile cavities were present in the vitreous. The stroma was uniformly infiltrated with lymphocytes. That the cavities were present in life was shown by the fact that they contained an albuminous fluid and cells. Indeed, on the wall of one of the spaces there were typical precipitates, while other aggregations of cells were caught free in the fluid. Here were present all the elements for the formation of precipitates, as E. Fuchs pointed out in connection with the anterior chamber, namely lymphocytes, cells that have the property of agglutinating, a fluid and motion. The pre-

cipitates of senile cavities are analogous to the precipitates of the anterior chamber. On the other hand, under other circumstances, cavities in old age may just as well contain pus cells.

(*b*) Red Blood Cells: Extravasations of blood in respect to the vitreous body take place either on its surface or into its stroma. In one

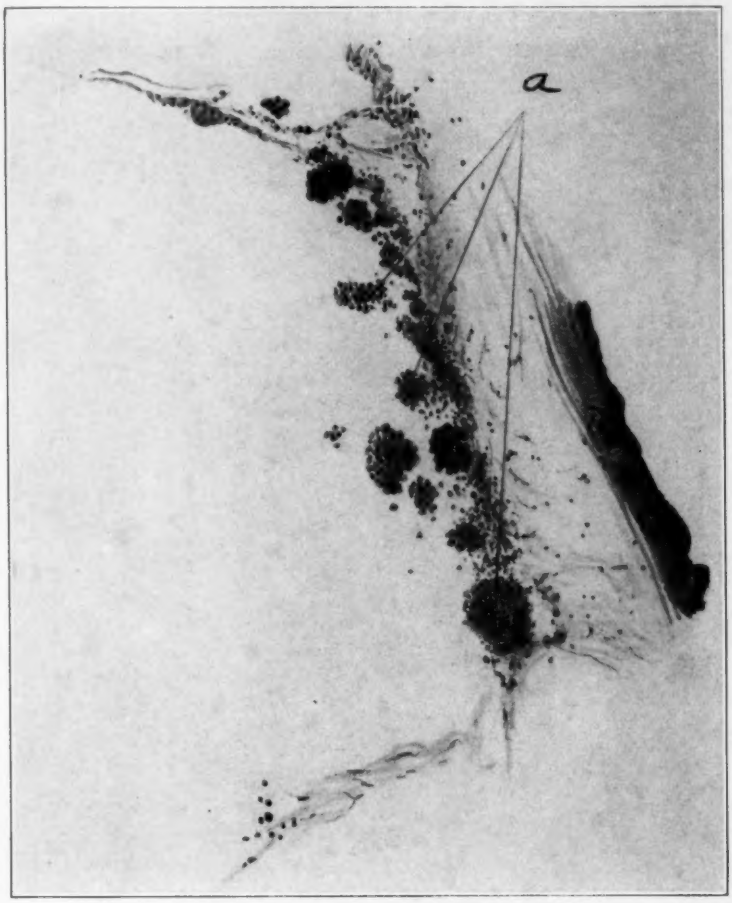

Fig. 5.—Detail of area shown in figure 4.

instance after injury the retrolental space was distended with erythrocytes. Anatomically this was a prehyaloid hemorrhage which in life simulated hemorrhage into the vitreous itself. It is remarkable that although the eye was enucleated seven months after the accident, the red blood cells in this space were in an almost perfect state of preservation. Similarly, blood confined between a detached Descemet's membrane and the parenchyma of the cornea may be preserved for a very long time.

In one case of a perforating wound there was a small hemorrhage in the macular region that lay compressed between the limitans interna of the retina and the posterior limiting layer of the vitreous. No other specimen so clearly revealed the two membranes as structures separate and apart. In other cases in the body of the vitreous hemorrhages were found confined to Cloquet's canal as they are sometimes surmised to be clinically. At other times the canal was free from blood while elsewhere the hemorrhages were collected in pockets in the stroma, generally lengthened out in the long axis of the eye.

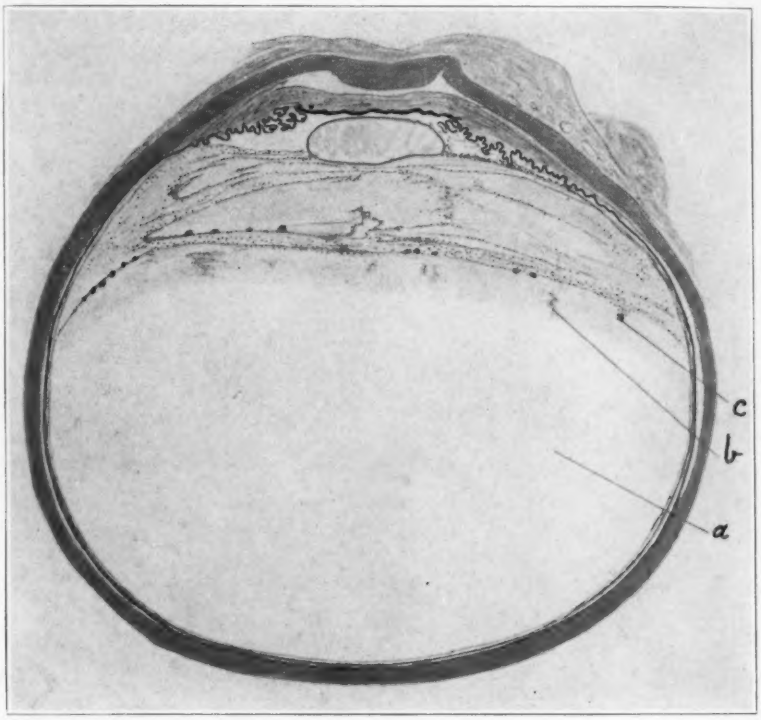

Fig. 6.—*a*, artificial detachment; *b*, true detachment; *c*, precipitates on the outer surface of the posterior limiting layer of the vitreous.

(*c*) Pigmented Epithelial Cells: 1. Intact. These cells may be driven bodily from the iris or the flat part of the ciliary body directly into the vitreous by injury. 2. Broken up. More frequently the cell walls are ruptured and the granules are set free in the vitreous. There were instances of both epithelial pigment and blood pigment in the same part of the vitreous. The granules from the epithelial cells are small and black, while those of blood pigment are larger, more irregular and brownish. The body of a macrophage packed with

granules from the pigment epithelium is difficult to distinguish from a pigmented epithelial cell. In either case the nucleus is hidden by the granules.

(*d*) Blood Vessels: Instances were seen in which blood vessels sprang directly into the vitreous covered only by their muscular coat.

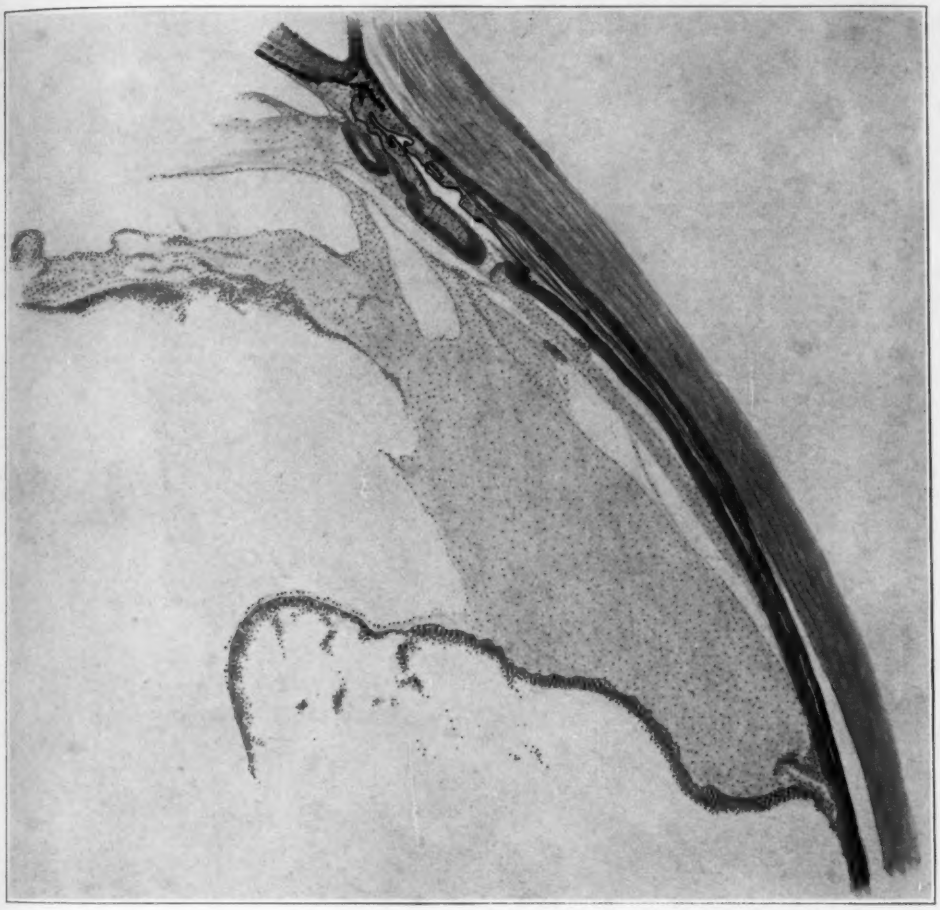

Fig. 7.—The posterior limiting layer is separated from the retina by a narrow zone of closely packed round cells.

No doubt, such naked vessels may be identified clinically. They arise from the vessels of the ciliary body, the retina or the optic nerve.

(*e*) Connective Tissue: Numerous eyes are enucleated because of secondary glaucoma as the result of obstruction of the central vein. The presence of hemorrhage and the interference with the circulation set up a chronic reaction that may lead to organization of the entire

vitreous. The new-formed tissue has a typical structure. There is always a central area of ordinary connective tissue with sparsely distributed nuclei and blood vessels. Covering this is a very cellular tissue composed of two elements: an inner layer of round cells and an outer one of elongated cells of endothelial character. While the main mass proliferates solidly into the vitreous, thin membranes spread from the sides over the adjoining retina.

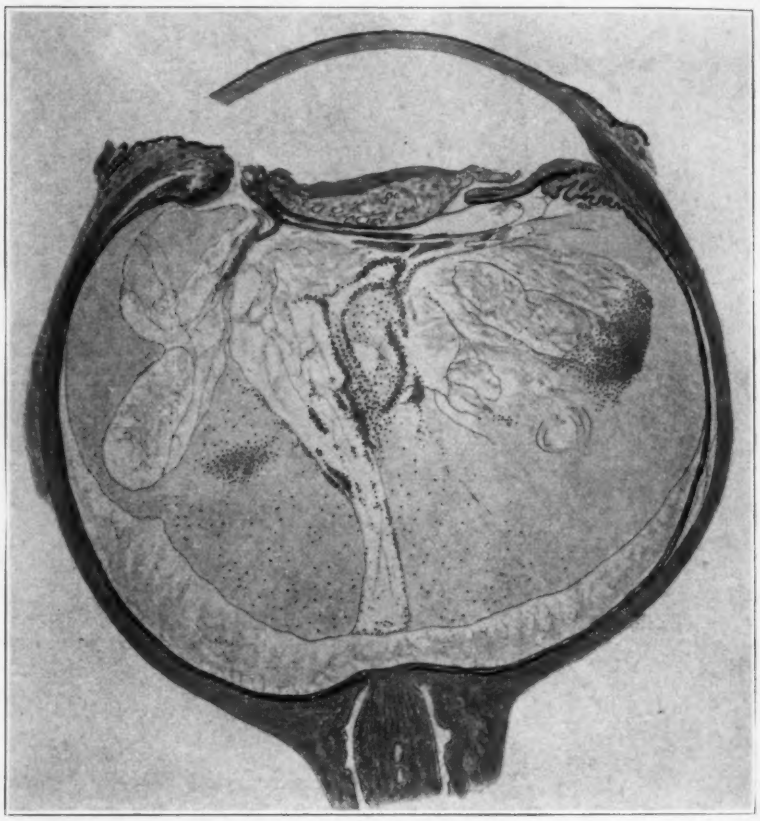

Fig. 8.—Detachment of the vitreous following infection after injury. Note the outlines of Cloquet's canal.

A flat whitish area in the fundus from under which the retinal blood vessels emerge may be explained by the presence of such a membrane. Some membranes are almost free from blood vessels; others are quite vascular with vessels of considerable size. A membrane may have a wide range over the retina. It is remarkable how well preserved the retina may be under it. These membranes lie so lightly on the retina that they are readily detached by transudation. When

detached they appear ophthalmoscopically as ballooned-out grayish membranes with blood vessels silhouetted against them. In cross-section under the microscope they suggest strands, from which they are distinguished by the fact that they confine a fluid—the transudate—which, being more albuminous than the fluid in the vitreous, takes a deeper stain with eosin. There are apt to be crops of membranes over the same area. Thus when one membrane is wafted off by transuda-

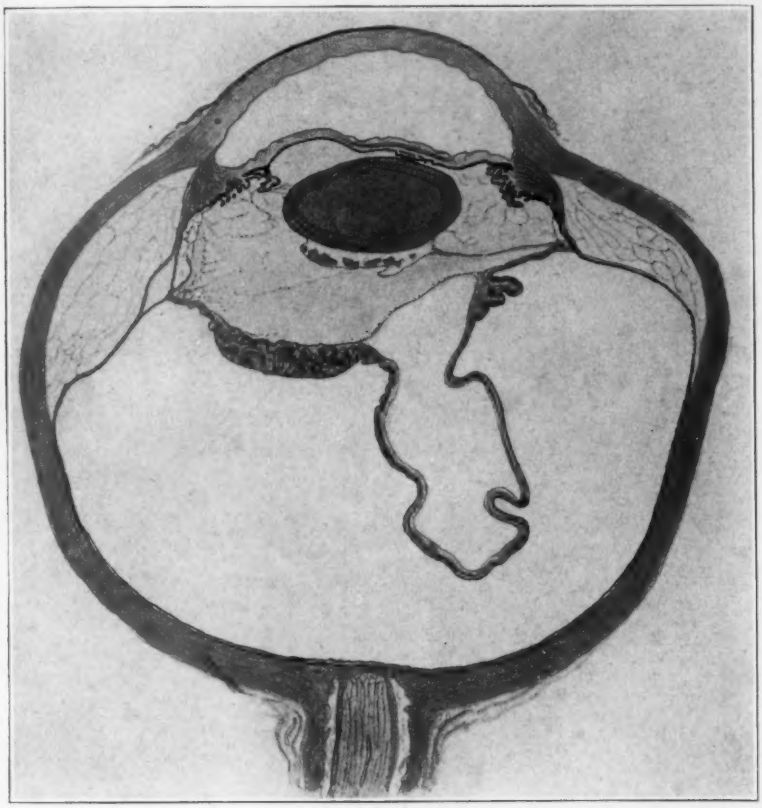

Fig. 9.—Cell sphere.

tion, another membrane forms, starting from the original exudative focus, so that superimposed membranes may extend into the vitreous. Owing to the pressure exerted by the underlying transudate and to their own tendency to contract, membranes bring about detachment of the retina. Occasionally membranes tear loose at their extremities and are wafted to and fro in the vitreous.

Massive exudates or hemorrhages undergoing organization are apt to assume a mushroom shape and are difficult to distinguish clinically

from growths. Jaeger published the report of a case of this kind in his day. One should study carefully the delicate membranes extending from all sides which when detached give the impression of being tensely inflated. In one instance of obstruction of the central vein, from a small focus of reaction that bound the retina to the choroid a delicate membrane originated that not only covered the retina, but

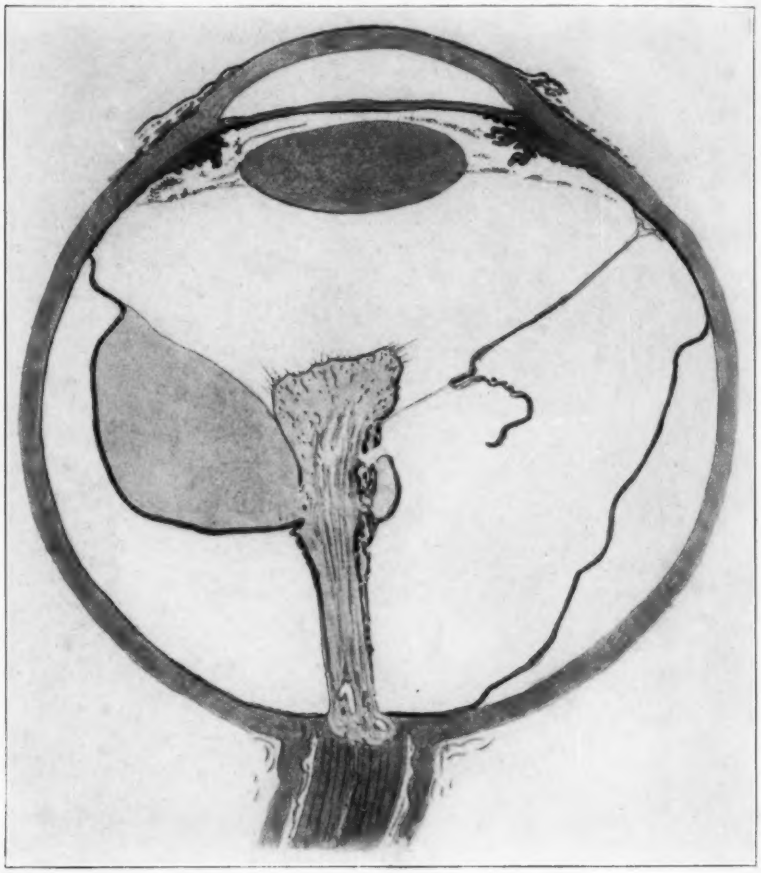

Fig. 10.—Tissue sphere.

even bridged over the deeply excavated nerve head. It is doubtful whether the excavation could have been seen in life, as there were too many nuclei and blood vessels in the membrane.

(*f*) Strands: From a primary focus of proliferation a solitary cord may shoot off into the vitreous to be whipped about in the fluid. Such are the strands in retinitis proliferans. The strands being wavy, the same section may pass through the same strand several times.

They have a central connective tissue core surrounded by a layer of round cells with a covering of flat cells. Strands are composed of the same elements as cyclitic membranes in general.

(*g*) Cell Spheres (Glial Spheres): Globular masses were encountered along the inner surface of the retina or on the nerve head. Sometimes these masses are free and at other times they are attached to the retina by a slender pedicle. Their origin can be traced to a

Fig. 11.—Coagulation of a degenerated vitreous around lymphocytes.

proliferation of the glial cells. There were instances in which similar spheres were seen on the external surface of detached retinas. These spheres have a covering of elongated cells resembling endothelium.

(*h*) Tissue Spheres without Pigment: Round bodies were found differing in origin from the previous ones. These consist of homogeneous masses thrown into folds between which are flattened cells. Other flat cells form the covering. They are entirely free in the vitreous chamber. They appear to be formed of remnants of broken-down coagulated stroma which, after being tossed about in a fluid

medium, become globular. Cells adhere to them at various stages of their development.

(*i*) Tissue Spheres with Pigment: Occasionally another variety of spheres was noted. These spheres contain pigment granules and are evidently products of the pigment epithelium that have worked their way through the entire thickness of the retina.

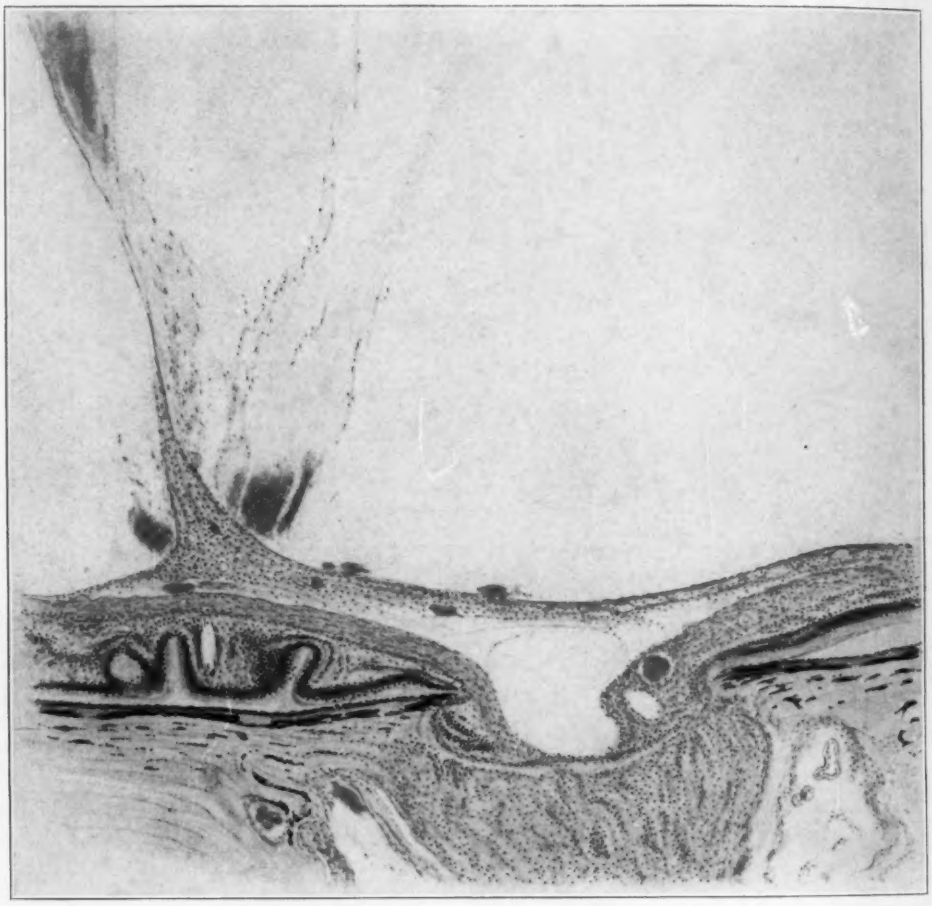

Fig. 12.—Fibroblasts in the vitreous filled with blood pigment.

OTHER CHANGES IN THE VITREOUS

It was interesting to note how exceedingly albuminous the compressed vitreous may be in cases of large sarcomas of the choroid. Thick clots of albumin were often encountered. In one case there was a coagulation of the fluid around the lymphocytes as if the mere presence of the cells had caused precipitation, just as one sees a saturated solution of sodium chloride precipitate around sand that is thrown in it.

SOLIDITY AND FLUIDITY OF THE VITREOUS

Cavity formation is so common in the aged that it may be considered physiologic. The usual situation of these cavities is well forward. Probably further back there is a general liquefaction of the vitreous, because here the stroma may not be dense enough to form walls. As far as vision is concerned, one can see as well through a fluid vitreous as through one that is gelatinous. The lamellae of the vitreous, when relieved from their connection with the central meshwork, fall back toward the periphery, where they adhere to one another to form denser layers. In this way the limiting layers of the vitreous may become thick and sharply defined. They were formerly referred to as hyaloid

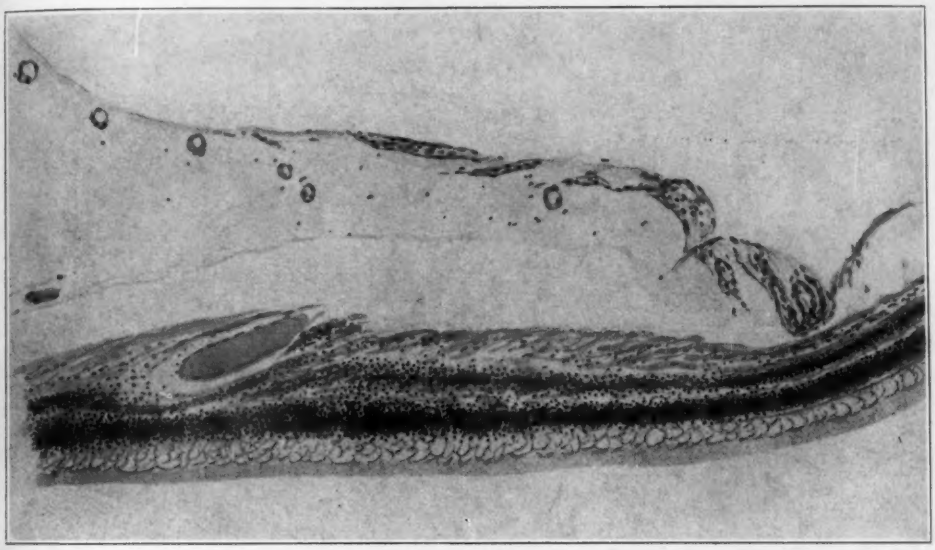

Fig. 13.—Separate detachments of choroid, retina and vitreous.

membranes. Usually the large wavy opacities seen clinically are situated just back of the lens, in the territory of the radiating lamellae which are normally thick.

In a case of high myopia in which an eye had been removed on account of carcinoma of the cornea practically nothing was left of the vitreous body but the thick fibrosed strands that came away from the base of the vitreous. In axial myopia and in staphyloma of the cornea the rule is an almost complete liquefaction of the vitreous. In such cases the anterior limiting layer is unusually thick and well outlined because of a fusion of lamellae. A dense anterior limiting layer stands one in good stead in the extraction of cataract, and it is in these cases of fluid vitreous that one may find this layer thickened.

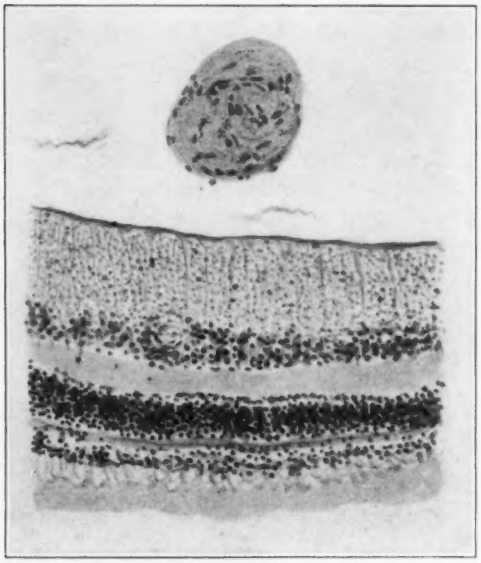

Fig. 14.—Delicate membrane with blood vessels easily detaching from the retina.

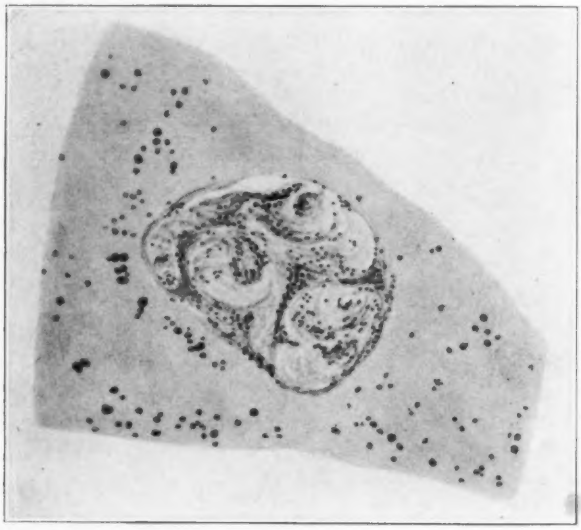

Fig. 15.—Membrane growing into the vitreous and covering over the excavated head of the nerve.

DETACHMENT OF THE POSTERIOR LIMITING LAYER OF THE VITREOUS

It is evident that the posterior limiting layer of the vitreous separates readily and frequently from the limitans interna of the retina. In detachment due to artefact the subhyaloid space contains a clear fluid taking no stain and free from cells. In true detachment there is always an albuminous fluid between the retina and vitreous body and there are apt to be cellular elements in it. The vitreous humour, having its fluid expressed, is all the more cellular because the cells occupy

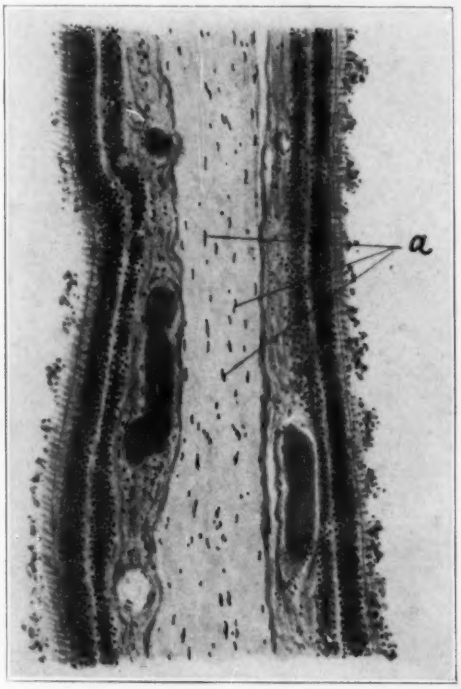

Fig. 16.—Dense funnel of connective tissue extending forward from the optic nerve, replacing the vitreous and detaching the retina.

less space. In life such a compressed vitreous would look correspondingly more cloudy.

(*a*) *In Relation to the Ora Serrata.*—It is only when the vitreous body is in a state of considerable liquefaction that the radiating lamellae are able to fuse and move about posterior to the lens. At the same time the posterior limiting layer may break up and strip off like wallpaper to join the lamellae in their excursions toward the center of the eye.

(*b*) *In Relation to the Optic Nerve.*—In a case of perforating corneal ulcer with a loss of the lens, the posterior limiting layer was

widely separated on all sides from the walls of the retina, but it remained anchored to the optic nerve from which it extended forward like a funnel to a point anterior to the equator of the eye. The nucleus of the vitreous had escaped with the lens, so that the peripheral lamellae were pressed to the center. In their folds were parallel lines of cells in the long axis of the eye, in conformity with the lamination of the stroma.

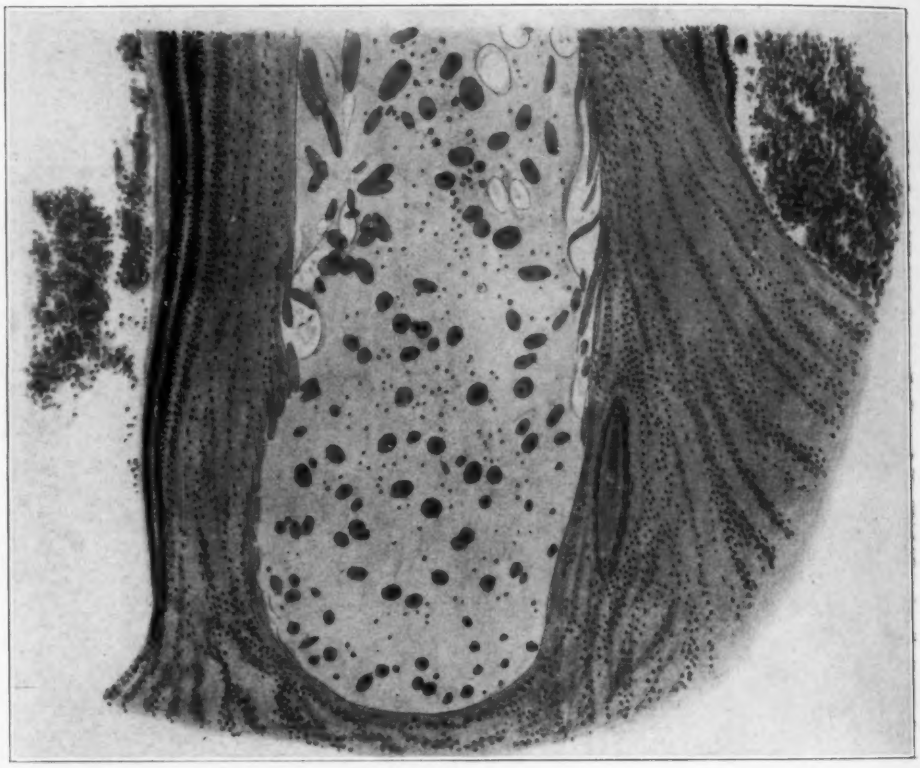

Fig. 17.—Detail of figure 16, showing the stroma bordered by a thick layer of cells of endothelial character.

In another case an eye was excised on account of glaucoma following spontaneous serous iritis. The posterior limiting layer was drawn back by a membrane into the center of an exceedingly deep excavation of the nerve head. It was separated from the limitans interna as far forward as the equator. The membrane was dense and sharply delineated, with precipitates on its outer surface. The posterior limiting layer, while rupturing elsewhere, may retain its anchorage at the nerve head. This would be an example of homogeneous membranes moving about in the vitreous far posteriorly. Cases were observed in which there was a complete tearing away of the posterior

limiting layer from the border of the nerve, resulting in a hole in this layer, a condition described by Sir William Lister and others. It is interesting that in all these cases of detachment the layer appeared denser than normal; otherwise it would have come away in fragments.

(*c*) *In Relation to Areas of Proliferation in the Retina.*—Where there are points of organization, as at the seat of a perforating wound, the layer may be bound down while detached elsewhere. In one sec-

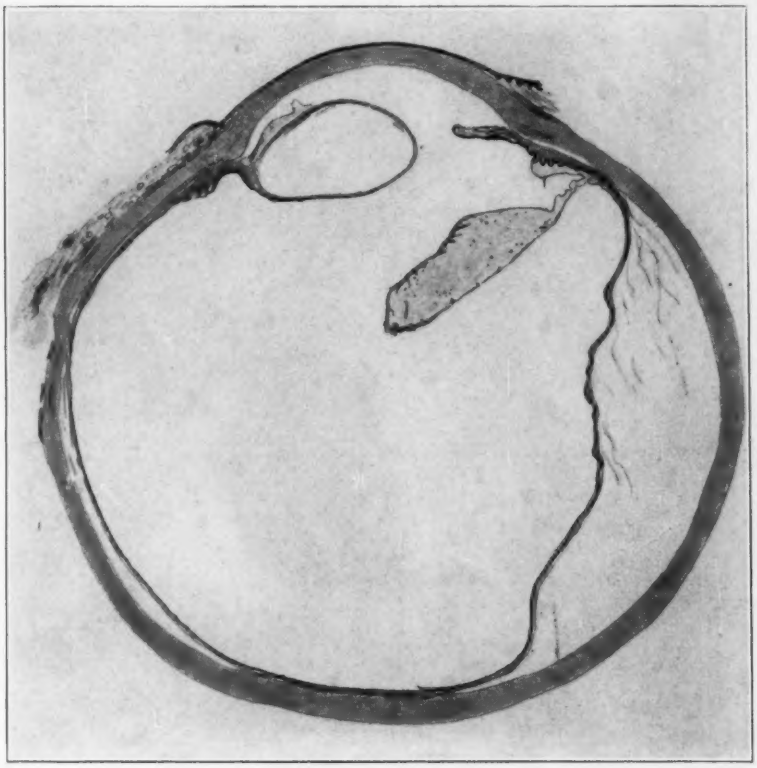

Fig. 18.—The vitreous is reduced to a small mass of connective tissue attached to the ciliary body by a pedicle.

tion the posterior limiting layer in detaching brought away with it a large excrescence that protruded into the vitreous from the retina. That it did not tear away at this point, producing a hole and leaving the excrescence behind, is explained by the fact that it was reinforced by a cellular membrane that spread over it from the base of the excrescence. In life this must have had the appearance of a translucent undulating membrane in the posterior part of the vitreous chamber, carrying with it a dense round opacity, the excrescence.

Instructive instances of detachment of the posterior limiting layers are associated with exogenous infection. The toxins thrown out in the vitreous by the bacteria that may be so severe that the layer is drawn up into welts or is warped like veneer in a very short time. It is often split up into *lamellae* by toxins, suggesting that after all it is but a modified part of the stroma.

One section shows a complete detachment of the posterior limiting layer which is drawn forward to the equator. At the same time there is a detachment of the anterior limiting layer which, being stripped from the zonular fibers and the lens, has fallen back almost to the

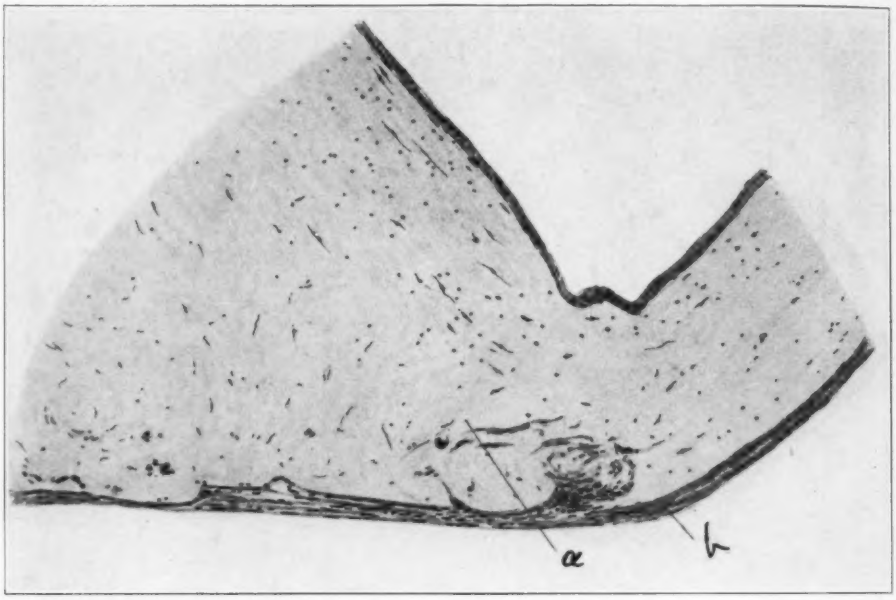

Fig. 19.—Detail of figure 18: *a*, the stroma is poor in nuclei; *b*, the border stands out by contrast, being rich in nuclei.

level of the ora serrata. In cross-section the vitreous appears as a broad band permeated with cells. It is certain that the anterior limiting layer does not become detached as easily or as often as the posterior layer. In many eyes after extraction of senile cataract it is notable that the anterior limiting layer exhibits a defect in the center leading into a wide and deep area of liquefaction in the vitreous. Doubtless some of the best results after cataract extraction in aged persons are those in which the anterior layer has been ruptured in the operation and thus elminated from the field of vision.

In all the preparations studied there was only one illustration of a tearing away of the base of the vitreous, and this was confined to one side. It occurred in a case of iritis with spontaneous dislocation of the

lens into the anterior chamber. The vitreous body was so contracted by organization that it resembled in cross-section an Indian club swinging by a cord from a thick layer of connective tissue that covered the ora serrata and the flat part of the ciliary body.

THE RÔLE OF VITREOUS OPACITIES IN DETACHMENT OF THE RETINA

Leber was the first to point out that it is striking how suddenly spontaneous detachment appears in myopia and in old age in eyes with normal refraction. Anatomically these two types of eyes are characterized by liquefaction of the vitreous with strands and lamellae oscillating from their attachment at the base. Leber expressed the belief, as did Gonin, that by traction these membranes produce rents in the retina just where it is thin and where it is likely to be friable in myopia and in old age. At the moment of the tear the already fluid vitreous passes over into the subretinal space. Gravity seems to play a part because it is superiorly that many detachments first develop, corresponding to the site where lamellae and membranes are most under the influence of gravity.

According to the sections, it is not alone in the immediate vicinity of the ora serrata that the posterior limiting layer is firmly attached. There seems to be a close union between it and the limitans interna of the retina almost as far back as the equator of the eye. This gives a broad zone of fixation. Tears in the retina at the anterior border of this zone may be ascribed to traction, exerted more particularly by the system of large radiating lamellae. These tears are impossible to locate clinically. At the posterior border of this zone the limiting layer may break up into plates and strands, which in their turn pull on the retina. The rents that one sees with the ophthalmoscope in the periphery of detached retinas originate in this region.

Whenever the posterior limiting layer is detached intact there proliferates on to its outer surface, from its point of anchorage to the retina, a cellular membrane similar to membranes encountered on the iris. One slide shows plainly how this membrane by contracting caused the retina to detach at the equator.

PROGNOSIS AS TO THE ABSORPTION OF OPACITIES

Cellular elements, such as erythrocytes and lymphocytes, when not too numerous, disappear without leaving any trace. It is possible that in time fine strands derived from the stroma may be dissolved while the grosser ones remain. Such products of degeneration as cholesterol crystals always remain. Connective tissue membranes and cords and spheres persist. All of these opacities that vanish do so spontaneously, so that it is difficult to judge the effect of treatment. Prof. Ernst Fuchs furnished material and many suggestions for the foregoing study.

OCULAR LESIONS OF RAT-BITE FEVER IN GUINEA-PIGS*

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AND
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Although the ocular lesions of animals with rat-bite fever (Sodoku) have an intrinsic interest, meriting study and description, the superficial resemblance of the eyes of guinea-pigs in the late stages of the disease to the eyes of horses affected with the periodic ophthalmia known as "moon-blindness" prompted us to investigate in detail these lesions in guinea-pigs. Since the beginning of this work, personal communications from Dr. Alan C. Woods, summarizing his unpublished studies of "moon-blindness" in horses, indicate that periodic ophthalmia of horses has nothing in common etiologically and little in common pathologically with the conjunctivitis, keratitis, uveitis and final panophthalmitis that occur in guinea-pigs infected with *Spirochaeta morsus-muris,* the organism causing rat-bite fever. Some of the original confusion concerning the condition was due to the incomplete descriptions of the ocular lesions of rat-bite fever that have been published. In order to supply lacking information and to describe a readily reproducible experimental disease of the eye as a part of a general infection, the results of our observations are recorded in this paper.

Ocular lesions are mentioned in many of the accounts of the natural and experimental rat-bite fever infections in man and in other animals. In the writings of the Japanese, as for example in the paper by Kusama, Kobayashi and Kasai,[1] swelling of the eyelids and conjunctivitis, with the presence of spirochetes in the lids, are described, without any specific mention of the lesions of the other structures of the eyes. Manson, in his "Manual of Tropical Diseases," mentioned the occurrence of keratitis during the course of rat-bite fever, and this has been pointed out again by Manson-Bahr.[2] A description of some

* Submitted for publication, July 21, 1930.

* From the Department of Bacteriology, School of Medicine and Dentistry, University of Rochester.

1. Kusama, S.; Kobayashi, R., and Kasai, K.: The Rat-Bite Fever Spirochete with Comparative Study of Human, Wild Rat and Field Vole Strains, J. Infect. Dis. **24**:366, 1919.

2. Manson-Bahr, P. H.: Manson's Tropical Diseases: A Manual of the Diseases of Warm Climates, ed. 8, New York, William Wood & Company, 1926, p. 198.

of the ocular lesions has been given by Theiler.[3] The review of rat-bite fever by Ruge[4] in Mense's "Handbuch der Tropenkrankheiten" contains a brief reference to inflammations of the eye, conjunctivitis, keratitis and iritis. The clearest descriptions of the ocular lesions of rat-bite fever have been presented by Mooser.[5] In fact, there is little besides our observations on lesions of the uveal tract to be added to his account of the condition. Mooser[6] was one of the first to note the similarity between experimental syphilis and the interstitial keratitis and other lesions of rat-bite fever in guinea-pigs and rabbits. These analogies with syphilis, which were apparently in the mind of Hata[7] in 1912, when he first advised using arsphenamine in the treatment for rat-bite fever, have been of especial interest to all students of the disease. They have recently been critically examined by Stühmer,[8] who in the course of describing rat-bite fever as a "model-infection" for studies of syphilis recorded the occurrence of ocular lesions in his infected guinea-pigs and rabbits.

KERATITIS IN STOCK GUINEA-PIGS

Guinea-pigs taken from supposedly normal stock have ocular lesions which might be overlooked if the eyes were not examined with a lens under proper illumination. In the course of selecting animals for use in these experiments, we found that about 30 per cent of the guinea-pigs had slight corneal lesions. These were small corneal ulcers, dryness and haziness of the cornea and linear or patchy opacities. Only one of the animals had a mild conjunctivitis with a small amount of mucopurulent discharge.

METHOD OF INOCULATION

The material used to infect the guinea-pigs in this series was citrated blood from guinea-pigs harboring *Spirochaeta morsus-muris*. Two strains of this spirochete were used. One known as the "Boston Strain" came from Theiler,[3] the other, known as the "Rochester Strain," was obtained from a patient suffering

3. Theiler, M.: Experimental Rat-Bite Fever, Am. J. Trop. Med. **6**:131, 1926.

4. Ruge, H.: Rattenbissfeiber, in Mense, C.: Handbuch der Tropenkrankheiten, Leipzig, Johann Ambrosius Barth, 1929, vol. 5, p. 621.

5. Mooser, H.: Experimental Studies with a Spiral Organism Found in a Wild Rat, J. Exper. Med. **39**:589, 1924; Experimental Studies with a Spiral Organism Found in a Wild Rat and Identical with the Organism Causing Rat-Bite Fever: II, ibid. **42**:539, 1925.

6. Mooser (footnote 5, second reference).

7. Hata, S.: Salvarsantherapie der Rattenbisskrankheit in Japan, München. med. Wchnschr. **59**:854, 1912.

8. Stühmer, A.: Die Rattenbisserkrankung (Sodoku) als Modellinfektion für Syphilisstudien, Arch. f. Dermat. u. Syph. **158**:98, 1929.

with rat-bite fever contracted in Rochester, N. Y. A description of this organism with an account of the experimental transmission of the disease to man was published by one of us (S. B.-J.[9]) in 1927. Except that the Rochester strain was somewhat less virulent than the Boston strain, there was no essential difference between the two. Both strains have been highly pathogenic for guinea-pigs and have invariably killed the guinea-pigs in from seventy to ninety days, or less, depending on the numbers injected. Each of the animals used for the study of the ocular lesions was given injections of 1 cc. of citrated blood from infected guinea-pigs, amounting to approximately 0.5 cc. of the undiluted blood. A somewhat heavy dose was given by injecting 0.5 cc. of the citrated blood subcutaneously and 0.5 cc. intraperitoneally. Twelve guinea-pigs, ranging in weight from 275 to 450 Gm., were given injections of the Boston strain of *Spirochaeta morsus-muris* and twelve were given injections of the Rochester strain.

RESULTS OF EXPERIMENTS

The guinea-pigs were kept under observation from thirty-three to seventy-eight days, with frequent examinations of their eyes. Most of the animals died of the infection or became moribund, when they were killed by etherization. A few were killed in the early stages to provide material for the study of the incipient lesions. All of the animals had some degree of swelling and crusting of the eyelids, associated with loss of hair around the eyes. In each group of twelve guinea-pigs, in seven (58.3 per cent) ocular lesions developed during these periods. Ten of the twenty-four guinea-pigs failed to show any significant disease of the eyes.

All of the animals went through the well known phases of experimental rat-bite fever. Spirochetes were found in the blood from thirteen to twenty-nine days after the inoculation. Ulceration of the skin of the jaw occurred in one animal. During the first period of from three to six weeks the guinea-pigs gained weight. At different times after this the animals began to appear sick: Hair fell off various parts of the body, particularly from the region around the eyes and the nose. The eyelids became swollen. Crusts formed on the edges of the eyelids. The ears became purplish and swollen, and the genitalia and anal regions became edematous, cyanotic and occasionally ulcerated. Most of the animals lost weight rapidly toward the end, losing as a rule about 50 Gm. in the last week of life. During the last stages of the disease, the animals were weak and dyspneic and ate little. The characteristic "facies" of rat-bite fever in guinea-pigs is shown in the accompanying drawing of an infected animal exhibiting corneal opacity (fig. 1). *Spirochaeta morsus-muris* was found in the conjunctival exudates of three of the animals.

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9. Bayne-Jones, S.: Rat-Bite Fever: Report of a Case, with Demonstration of the Causative Organism and Its Use in Treatment of Paresis, New York State J. Med. **27**:1113, 1927.

OCULAR LESIONS; APPEARANCE IN LIVING ANIMAL

As the gross and microscopic appearance of the ocular lesions were almost identical, varying somewhat in degree, in all of the fourteen animals exhibiting them, the following protocols have been chosen to serve as illustrations of this phase of the disease.

PROTOCOL 1.—Guinea-pig 123: Inoculated March 13, 1928, with 1 cc. of citrated blood from guinea-pig 74, carrying "Boston Strain" of *Spirochaeta morsus-muris*. Weight 350 Gm. Very slight linear opacity of cornea of left eye at the left pupillary border. Right cornea clear.

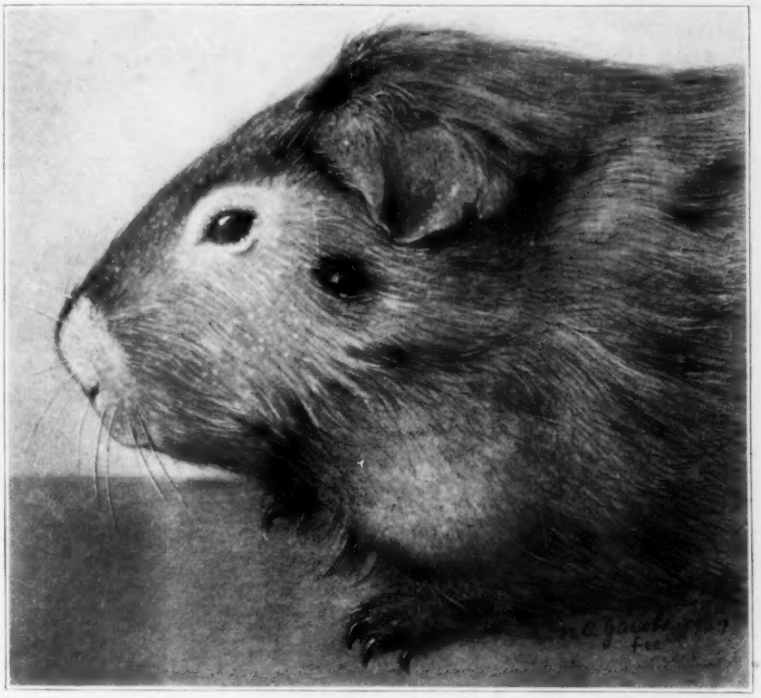

Fig. 1.—Facies of rat-bite fever in the guinea-pig, showing alopecia around the eyes and jaw, ulcer on the skin of the jaw, conjunctivitis with exudate and crusts on the swollen lids and opacities and lack of luster of the cornea.

March 26: Spirochetes in blood. No change in eyes. Weight, 360 Gm.

April 11: Spirochetes in blood. Weight, 395 Gm. Left eyeball injected, and same small corneal opacity present. Right eye showed mild injection and lacrimation, cornea clear.

April 24: Weight, 419 Gm. Left cornea dull. Right cornea had a deep central opacity and the rest of the cornea was dull. The conjunctiva of both eyes were injected. Eyelids swollen. Keratoconjunctivitis of both eyes.

May 8: Weight, 395 Gm. Left cornea showed cloudiness, vascularization and central opacity. Evidence of interstitial keratitis. Right cornea had lost transparency. The central opacity was not visible.

May 22: Weight, 455 Gm. No spirochetes found in blood or in conjunctival exudate. Left eye showed conjunctivitis and keratitis, with central opacity. Right cornea showed three small opacities. Lids swollen. Alopecia present.

June 1: Weight, 430 Gm. Condition of eyes as noted. Killed. Eyes removed for section.

PROTOCOL 2.—Guinea-pig 127: Inoculated March 13, 1928, with 1 cc. of citrated blood from guinea-pig 74; "Boston Strain." Weight, 348 Gm. Both eyes normal and corneas clear.

March 26: Spirochetes in blood. Small, streaky, linear opacity in left cornea. Right cornea clear. Weight, 363 Gm.

April 11: Weight, 382 Gm. Same linear opacity in left cornea with cloudiness and injection of the limbus. Cloudiness and injection of the upper portion of the right cornea.

April 24: Weight 369 Gm. Swelling of lids. Lacrimation and scaliness of lids. Corneas same as on previous examination.

May 8: Weight, 388 Gm. Left cornea entirely cloudy, with densest opacity at the margins. Conjunctivitis and swelling of lids. Right cornea cloudy.

May 22: Weight, 420 Gm. Right cornea showed a deep central opacity. Left cornea showed a crescentic opaque ulcer, ulceration and deep infiltration, and definite keratitis. No spirochetes found in the conjunctival exudate.

June 1: Weight, 398 Gm. Opacities of both corneas. Swelling of lids, alopecia and conjunctivitis. Killed, and eyes removed for section.

PROTOCOL 3.—Guinea-pig 137: Inoculated March 13, 1928, with 1 cc. of citrated blood from guinea-pig 104, carrying "Rochester Strain." Weight, 421 Gm. Both eyes clear, entirely normal.

March 26-27: Spirochetes found in blood. Weight, 440 Gm. Both eyes clear.

April 11: Weight, 434 Gm. Spirochetes in blood. Right eye showed lacrimation, swelling of lid and diffuse opacity of the cornea. Left eye showed scaly border of the lid, cornea clear.

April 24: Weight, 442 Gm. Left cornea clear, with slight injection at the limbus. Scaliness of lids. Right cornea clear except for small opacity.

May 8: Weight, 408 Gm. Left cornea clear. Lacrimation. Right cornea showed small facet opacity. Blepharitis and loss of hair around both eyes.

May 22: Weight, 407 Gm. Small opacities in both corneas. Conjunctivitis present. Spirochetes (*Spirochaeta morsus-muris*) found in conjunctival exudate.

May 23: Died.

These animals, like others in the series, showed evidence of interstitial keratitis, with occasional ulceration of the cornea. The corneal cloudiness and vascularization began about two or three weeks after the inoculation. In some it progressed to a general opacity of the cornea; in others the corneal opacity varied in density from time to time, and the positions of the small opacities changed. In this respect the corneal lesions tended to have a periodicity, being more intense at some times than at others in the same animal. No flakes or deposits were seen in the anterior chambers of these eyes with the ordinary methods of illumination and the Bausch and Lomb hand slit-lamp, and although the microscopic study of sections showed that the iris was more or less infiltrated, these changes were not noted when the eyes were examined in the living animals.

After the removal of the eyes from dead animals, they were fixed in Zenker's fluid for paraffin embedding and sections for hematoxylin and eosin stains and in a dilute solution of formaldehyde, U. S. P. (1:10) for Levaditi preparations.

The question of the occurrence of *Spirochaeta morsus-muris* in the corneal lesions of animals with rat-bite fever appears to have been settled definitely by Mooser[10] and a number of other investigators, who have found spirochetes in these locations in tissue stained by Levaditi's method. In a careful search through many sections of our material we have found darkly stained curved structures resembling this spirochete between the corneal lamellae, in the region of the small vessels, and in the anterior layers under Bowman's membrane. The spirochete,

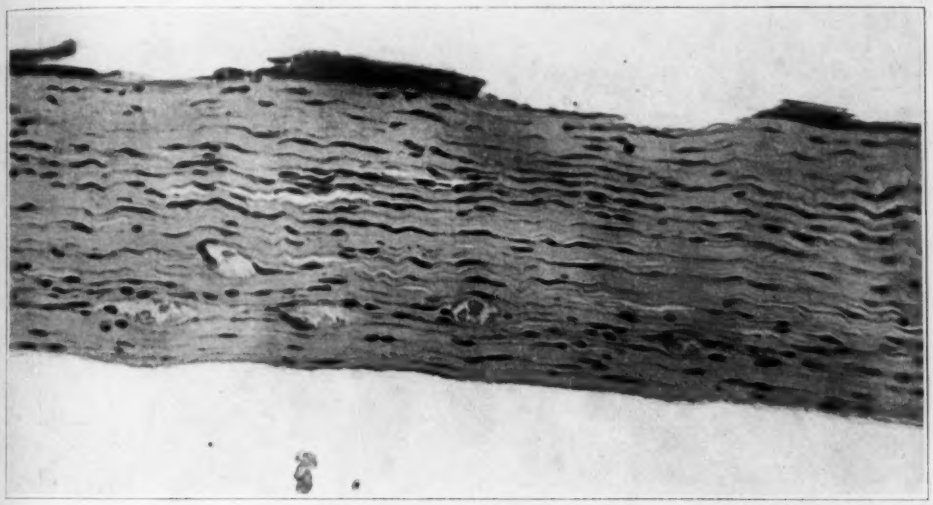

Fig. 2.—Section of the cornea from guinea-pig 127, showing two small ulcers, with loss of epithelium down to Bowman's membrane and probable break in this membrane. There are capillaries in the posterior layers of the substantia propria, with slight cellular infiltration.

however, is so small and the chance of deposits of silver mimicking the organism so obvious that interpretation of the observations is difficult. The organisms probably occur in these corneal lesions, but are not numerous.

OCULAR LESIONS; HISTOPATHOLOGY

Sections of the affected eyes and ocular adnexa exhibit the following pathologic changes:

Lids.—In some cases the epithelium appears to be thickened; in others there is a loss of several layers of epithelial cells at the margins

10. Mooser (footnote 5, first reference).

of the lids, producing small pitlike ulcers. The connective tissue is edematous and infiltrated with round cells, plasma cells and polymorphonuclear leukocytes. There are groups of round cells surrounding small blood vessels. The appearance is that of a diffuse blepharitis. The hair follicles are surrounded by groups of round cells, plasma cells

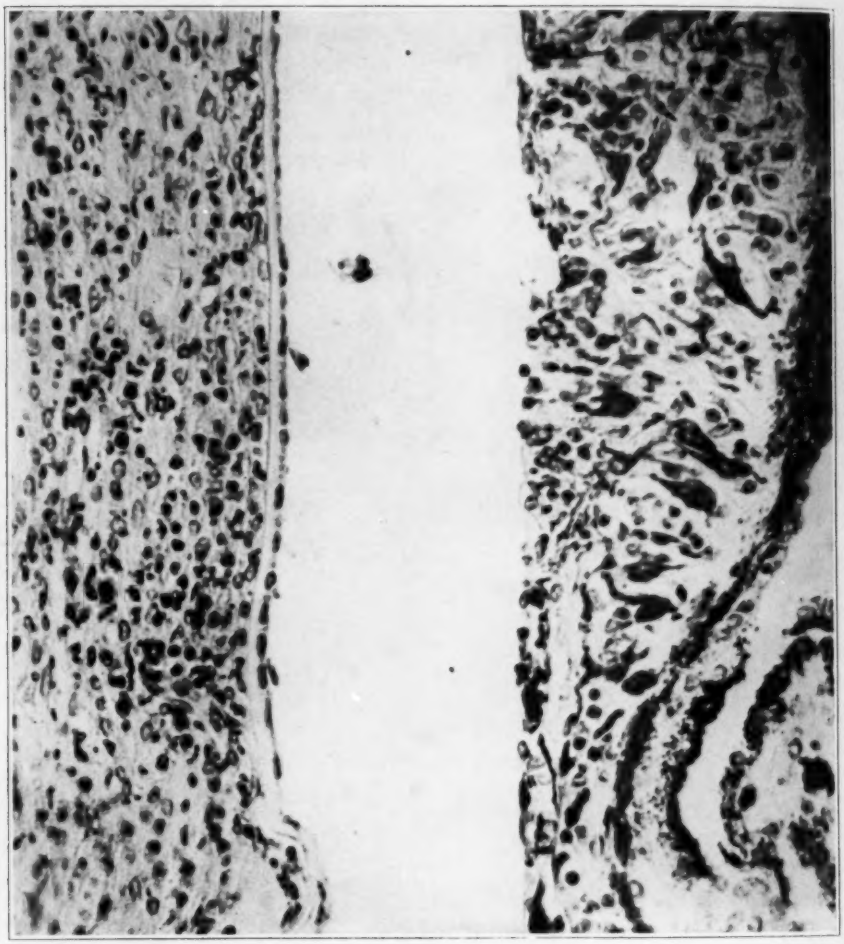

Fig. 3.—Section through the posterior edge of the cornea and the iris. There is infiltration with round cells and a few polymorphonuclear leukocytes in the layers of the cornea and involving the region of Descemet's membrane. The posterior endothelium of the cornea is intact. There is moderate infiltration of the iris with round cells and plasma cells (guinea-pig 123).

and a few polymorphonuclear leukocytes, but the epithelium is essentially normal, and they present no evidence of acute folliculitis. An anatomic basis for the alopecia is not apparent. The meibomian glands, vessels and muscles of the lid are normal.

Conjunctiva.—The epithelium of the palpebral conjunctiva is vacuolated and occasionally infiltrated with both round cells and polymorphonuclear leukocytes. Groups of these cells occur around numerous small vessels in the subconjunctival tissue. Similar groups of cells, together with diffuse infiltration with round cells, polymorphonuclear leukocytes and plasma cells, are seen in the outer zone of the bulbar conjunctiva. The degree of conjunctivitis varies with the stage of the

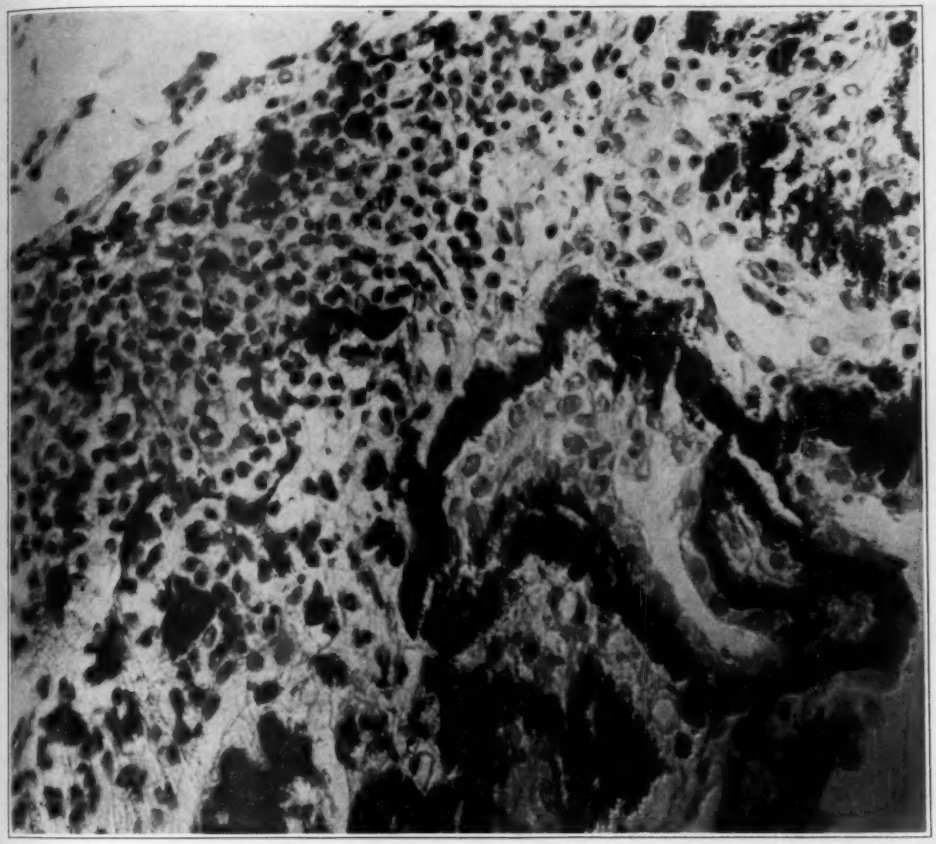

Fig. 4.—Section through the ciliary process of the same eye of guinea-pig 123, showing the cellular reaction in the ciliary process.

disease. It is most intense in the late stages when secondary pyogenic organisms invade the conjunctiva.

Sclera.—Except for groups of round cells and plasma cells around some of the vessels in the outer layer of the sclera, this structure shows no pathologic condition.

Cornea.—The epithelial cells are swollen, pale and vacuolated in some areas of the cornea. No inclusions were recognizable in these

cells. Solution of continuity varying from simple exfoliation to ulceration is shown in the series of sections. The small ulcers reach Bowman's membrane and occasionally penetrate to the substance of the cornea, as shown in figure 2. Small blood vessels, sometimes accompanied by cellular infiltration, occur frequently in the posterior layers of the cornea. In several sections these vessels are numerous also in the anterior layers. The corneal lamellae are often separated and swollen. A dense infiltration with round cells and polymorphonuclear leukocytes is found in the region of the corneoscleral junction. In this location the foreign cells are in contact with the posterior limiting membrane, suggesting a descemetitis. Descemet's membrane, however, is usually intact, and the covering endothelium is not abnormal. This is shown clearly in figure 3.

Ciliary Body and Iris.—Round cells, plasma cells and a few polymorphonuclear leukocytes infiltrate the ciliary body and the iris. The epithelium covering these structures remains intact. No exudate or fibrous adhesions occur on the surfaces of these structures. The iritis and cyclitis appear to be moderate, without producing fibrinous exudate or leading to fibrous adhesions.

Choroid.—No significant abnormality occurs in this layer of the eye, aside from occasional groups of round cells in the perivascular tissue. Choroiditis is negligible.

Retina and Optic Nerve.—No lesions are seen in these structures.

Lacrimal Gland.—There are dense infiltrations of round cells, plasma cells and a few polymorphonuclear leukocytes in the tissue around the ducts. In a few areas, linear and triangular extensions of this cellular infiltration pass between the acini of the gland.

External Ocular Muscles and Orbital Fat and Connective Tissue.—Similar infiltrations of cells are associated chiefly with the blood vessels.

The sections of the lens were not satisfactory. Fragments of the lens in these sections appeared to be normal.

SUMMARY AND CONCLUSIONS

Lesions of the eyes and contiguous structures occurred in more than half (about 60 per cent) of the guinea-pigs with experimental rat-bite fever, produced by infection with *Spirochaeta morsus-muris.* The infection is reproducible with regularity in these animals and provides a convenient method for the investigation of ocular lesions occurring in the course of a systemic infection.

These lesions vary in intensity during the course of the disease, but are generally severe at the time when the animals are moribund

with the infection. The infection produces blepharitis, conjunctivitis, interstitial keratits with corneal ulceration and moderate iritis and cyclitis. Characteristic cellular infiltrations occur also in the lacrimal glands, chiefly around the ducts, and in the perivascular regions in the external ocular muscles.

Spirochetes were found in the conjunctival exudate, but could not be demonstrated with certainty in the cornea.

THE RATIONALE OF THE DENIG TRANSPLANT IN TRACHOMA

WITH MICROSCOPY OF THE GRAFT IN TWO CASES *

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The Denig graft was designed by its author to check the most serious sequel of trachoma, the formation of pannus. His object was to interpose between the cornea and the diseased conjunctiva of the fornix a stratum of tissue that the infection could not bridge. He asserted that by transplanting at the upper limbus a section of the mucous membrane of the mouth, a pannus could be checked or even ameliorated. The

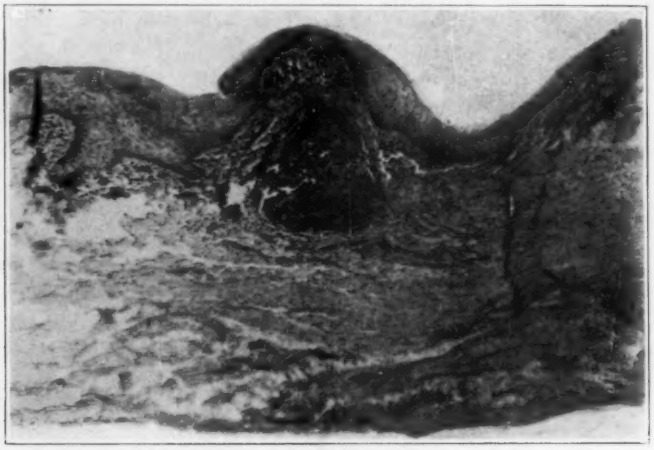

Fig. 1 (case 1).—Formation of follicle and thinning of overlying epithelium.

operation was based mainly on the premise that the mucosa of the mouth was proof against the invasion of trachoma.

Partly to verify the truth of this premise and partly to improve the cosmetic results of the grafts, sections were taken from the transplants of each of two patients who had previously been operated on according to Denig's method.

EXAMINATION OF SECTIONS

The removal of the sections in both cases was done one and one-half years after the operations. The lids were in a fairly good condition; it was not possible, however, to say that no latent trachoma was present. There were no follicles or trichiasis at the time of the Denig operation. In one case a folliculosis in the

* Submitted for publication, July 11, 1930.

* From the State Eye Hospital, Budapest, Prof. Joseph Imre, Jr., Director.

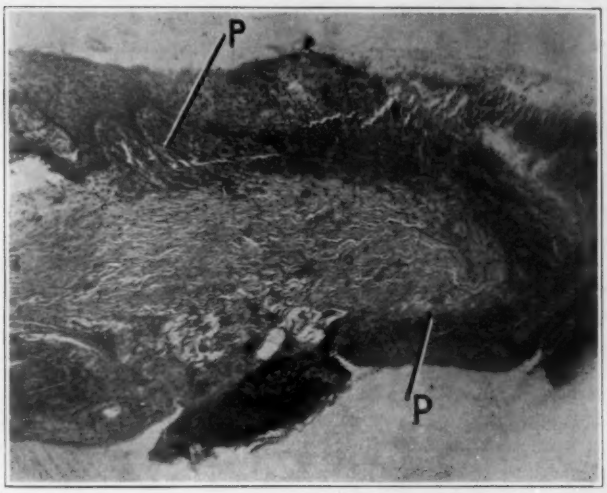

Fig. 2 (case 1).—Dense infiltration, chiefly of plasma cells (*P*).

Fig. 3 (case 2).—This section is curled on itself. Note the extreme vascularity of the subepithelial tissues; lymphocytic infiltration.

graft was seen grossly, for which a scraping and expression had previously been performed. The other graft was flat and gave no indication clinically of its microscopic pathology.

Histologic examination revealed typical trachomatous infection of both grafts, each representing one of the two chief types of trachomatous changes: one showing the formation of follicles and the other a diffuse infiltration.

Microscopic Observations.—The center of the follicle contained a mass of epithelioid cells, most of them in a state of degeneration. Fragments of disintegrated débris were seen in the fine connective tissue stroma. The outer rim of the follicle consisted mostly of dark-staining small lymphocytes. Numerous blood vessels around the follicle sent branches toward the interior. The epithelium directly over the follicle was markedly thinned. In another area of the same section the subepithelial spaces were densely packed with plasma cells, a few lymphocytes and still fewer polymorphonuclear leukocytes. Many of the plasma cells showed granular degeneration and stained faintly.

The other section showed no folliculosis; there was only a high degree of vascularization of the subepithelial tissues, with a marked infiltration of small mononuclear leukocytes. Many of these lymphocytes showed pyknosis and cellular degeneration indicative of a severe chronic infection. Such degenerative changes were here more evident than in the sections showing the follicles.

In both cases the limbal as well as the conjunctival borders of the sections were involved. No inclusion bodies were found in the scrapings from either case.

COMMENT

Denig stated that the rationale of his transplant is founded on the following assumptions: 1. It acts as a barrier against trachoma from the scleral conjunctiva. 2. It improves the nutrition of the cornea, qualitatively. 3. It increases the nutritive fluids to the cornea, quantitatively. 4. It protects the cornea to some extent from the rubbing of the lids.

It is known that the trachomatous process can pass through the intervening mucosa and advance toward the cornea. Seefelder, in 1928, showed the same condition in one of his cases. He pointed out that one must bear in mind Denig's postulation: The lids must be free from follicles or trichiasis before one attempts the operation.

Denig inferred that the corneal nutrition must suffer as the result of pathologic changes in the vessel walls. If this is so, it is difficult to understand how nutrition of the cornea can be improved qualitatively by the ingress of new vessels which, in the two cases mentioned, are obviously themselves surrounded by trachomatous infiltration. Their walls must suffer just as certainly as those of the original vessels of the bulbar conjunctiva.

Nor is it clear how the nutritive fluids to the cornea are considerably augmented in volume. How does this quantitative increase take place? The nutrition of the cornea is normally derived from two sources: the rich network of conjunctival vessels that fringes the limbus, and the sparser twigs given off more deeply by the anterior ciliary arteries. In

spite of the paucity of the latter they are sufficient to maintain the corneal nutrition, for if one dissects away the conjunctiva from even the entire circumference of the healthy cornea no sloughing follows. The trachomatous cornea cannot have a different vascular supply. What is the origin of the vessels of the pannus, and how are they affected by the resection of the limbal conjunctiva and the interpolation of the graft? Ichikawa showed that the pannus is the result of the spreading of the trachomatous process from the bulbar conjunctiva to the cornea, and that it consists of two chief elements: the cellular (small round cells and young connective tissue cells) and the vascular (newly formed vessels). From Ichikawa's observations, one might expect a severance of the blood supply to the pannus during the transplantation operation. But this does not take place. If one observes carefully when the conjunctiva adjoining the limbus is cut, one detects no blanching of the vessels. The vessels, then, must have their major origin in the episcleral or scleral tissues, that is, from the anterior ciliary arteries, and are not affected by the transplantation operation. If one considers these anterior ciliary branches in the pannus as possessing nutritive possibilities, one must assume that they pass more than sufficient foodstuffs to a cornea that ordinarily must have a far lesser supply, since blood vessels are not naturally found in the stroma.

Any increment in nutritive fluid resulting from the grafting, then, must come from the graft itself. It is possible for the graft to become spontaneously absorbed without altering the appearance of the pannus. Considering the vascularization of the cornea as an endeavor on the part of nature to better the nutrition of this tissue, it would seem that since the graft absorption does not change the vascularity of the cornea, the graft has little to do with the corneal nutrition. How much added nutritive material the implant can pass into the cornea, whether this extra nutrition is really needed in view of the already abundant supply, and how much of therapeutic assistance is thereby rendered are matters of speculation. It is my opinion that the corneal involvement is not one of faulty nutrition with its attendant degeneration, but rather a response to the stimulus of infection, and that if the cornea clears up at all, it is only because the virulence of the infection has abated.

Regarding the mechanical protection afforded by the graft, it is conceivable that the thick implant raises the lid away from the limbus and to some slight degree decreases the friction from a roughened lid.

In removing a section from a Denig graft, one must know (1) that the bleeding is profuse and annoying but readily controlled by two or three sutures passed through the wound margins, and (2) that the procedure is painful beyond expectation, requiring, besides the instillation of cocaine, an injection of procaine hydrochloride into the graft.

SUMMARY

1. The mucous membrane of the mouth employed as a perilimbal graft is not immune to the invasion of trachoma.

2. The vascularity of the pannus is apparently not altered by the removal of the limbal conjunctiva or the substitution of the graft.

3. The relation between the solution of the pannus and the "improved nutrition" of the cornea is speculative.

It has not been my purpose to pass judgment on the clinical efficacy of the Denig graft, but merely to inquire into the soundness of its theoretical principles.

THE FREQUENCY OF ASTIGMATISM*

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AND
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Since the introduction of the ophthalmometer into clinical ophthalmology, many authors have studied the variations of the curvature of the cornea. Steiger, who was the first to apply statistical methods to this study, found that the variations of the corneal curvature behaved very regularly and followed the law of Gauss. The next logical step was to investigate the distribution of the various degrees of corneal astigmatism over the three main refractive states, emmetropia, myopia and hyperopia. This classification, mainly on Steiger's suggestion that all the refractive states should be considered as physiologic variations of normal refraction, was learned to be an arbitrary procedure which was not justified by any biologic facts. Therefore, the frequency of the various degrees of corneal astigmatism was determined separately for each refractive state. Extensive studies of this type, using skiascopy without cycloplegia and manifests for the determination of axial refraction and then correlating these data with the corneal astigmatism, were made by Menestrina.[1]

All the work thus far considered only the corneal astigmatism. In the early days of ophthalmology it had been shown by Donders,[2] Knapp, Javal and Gullstrand that even for purely practical purposes it was necessary to assume an optical asymmetry of the lens. Up to date few actual measurements of meridional asymmetries of the lens have been made. Thomas Young was able to show on his own eyes that after neutralizing the corneal asymmetry by submerging his eyes under water, an inverse astigmatism of 1.7 diopters was present. Similar studies were carried out by Schioetz and Tscherning. The determination of the curvature of the surfaces of the lens is still a complicated and time-consuming task, and at the present time, no large quantity of material is available for statistical purposes. The figures

* Submitted for publication, June 30, 1930.
* From the Eye Clinic of the University of Chicago, E. V. L. Brown, Director.
* Read before the Chicago Ophthalmological Society, May 19, 1930.

1. Menestrina, G.: Astigmatismo corneale ed ametropie assili, Arch. di ottal. **33**:399, 1926.

2. Donders, F. C., quoted by Hess (footnote 9).

recently published by Tron [3] cannot be given full credit because of the incorrectness of Tron's optical and mathematical methods. The posterior surface of the cornea also was found asymmetrical by Tscherning, and recently by Nicoletti.[4] Whatever the importance of each of these components might be, it was made clear that the so-called total astigmatism differs from the corneal astigmatism to such an extent that no constant relation between the amount and the axis of the two kinds of astigmatism can be given. The formula which Javal gave as a means of figuring the total astigmatism after measuring the corneal asymmetry is known to be decidedly inaccurate and unreliable.

It is our contention that for statistical purposes the only present accurate way of determining the frequency of total astigmatism is by retinoscopy of a sufficiently large number of eyes, which—at the time of retinoscopy—were under the influence of a complete cycloplegia. It may still be a subject of discussion whether or not the corrections found under these circumstances should be prescribed. But there can be no doubt that for the purpose of statistical studies all the persons examined must be under the same conditions.

A complete survey of the literature revealed no previous work along this line. We are aware of the fact that our observations cannot be compared directly with those of any other authors. We herewith present our material as the first concerning the frequency of total astigmatism.

STATISTICAL DATA

The data for this paper were obtained from a study of the same 10,000 records from the office practice of E. V. L. Brown that were reviewed for the paper entitled "The Refraction Curve in the United States," written by Brown and Kronfeld, and delivered at the International Ophthalmological Congress in Amsterdam in September, 1929. As in the previous paper, only atropinized eyes, excluding all those with corneal and lenticular diseases, were selected. From this group of eyes we have used only those that showed 0.50 diopters or more astigmatism, and have considered those eyes with 0.25 diopters or less as nonastigmatic.

For the purpose of recording, we arranged our astigmatic cases as shown in table 1. This table represents a coordinate system in which the spheres are indicated on the abscissa and the corresponding cylindric components on the ordinate. The figure found in each unit of the coordinate system indicates the number of cases that required the respective

3. Tron, E.: Variationsstatistiche Untersuchungen über Refraction, Arch. f. Ophth. **122**:1, 1929.

4. Nicoletti, G.: The Influence of the Posterior Surface of the Cornea on the Total Astigmatism, Ann. di ottal. e clin. occul. **55**:987, 1927.

spherocylindric combination. The majority of cases required minus spheres combined with minus cylinders (the right inferior quadrant of the table) and plus spheres combined with plus cylinders (the left upper quadrant of the table). In addition, a considerable number of cases were found that needed a combination of a minus sphere with a plus cylinder (the right upper quadrant) or a combination of a plus sphere with a minus cylinder (the left lower quadrant).

For statistical purposes it was necessary to present the material as a continuous row. This meant that the actual amount of astigmatism should be expressed in terms of plus cylinders or minus cylinders. We carried out both procedures. The former, using plus cylinders only, was designated procedure A, for which it was obligatory that all our spherocylindric combinations be transposed into combinations of a sphere with a plus cylinder. Through this process of transposition, cases were listed on the abscissa with the value of their stronger meridian. The logical result of this procedure (A) was that the astigmatic eyes as a whole came to lie more toward the myopic side of the coordinate system than in procedure B, in which every case was expressed as a combination of a sphere with a minus cylinder. In other words, in procedure A the stronger meridian of the astigmatic system determined the place on the abscissa where the case should be listed, while in procedure B it was the weaker meridian. For the purpose of comparing the astigmatic cases with the entire material of Brown and Kronfeld (astigmatic plus nonastigmatic eyes), it was easy to see that neither procedure A nor B was correct. A shifted the relative frequencies too far toward the myopic side, while B led to a similar mistake toward the hyperopic side. This is shown in tables 2 and 3. Table 2 compares the results of procedures A and B, while table 3 presents the relationship for each specific refractive state between A and B and the total number of astigmatic plus nonastigmatic eyes. This comparison contains a considerable error, which is due to the fact that the figures for the astigmatic eyes are taken from table 2 and are approximately correct. The total number of eyes in the material of Brown and Kronfeld is not correct and cannot be given correctly, because these authors listed their cases with the main meridians as the unit of refraction. The figures that were obtained by dividing the data of Brown and Kronfeld by 2 give only approximately the number of eyes, because in the majority of instances the two main meridians of an eye did not fall into the same group. This explains why in some of the groups more astigmatic eyes were found than there were astigmatic plus nonastigmatic eyes. This error, however, may be negligible for the time being, and we can conclude from table 3 that in groups I, V and XV to XIX there are practically no nonastigmatic eyes, while in groups VI to XIV a certain percentage of nonastig-

TABLE 1.—*Astigmatic Cases Arranged According to Groups**

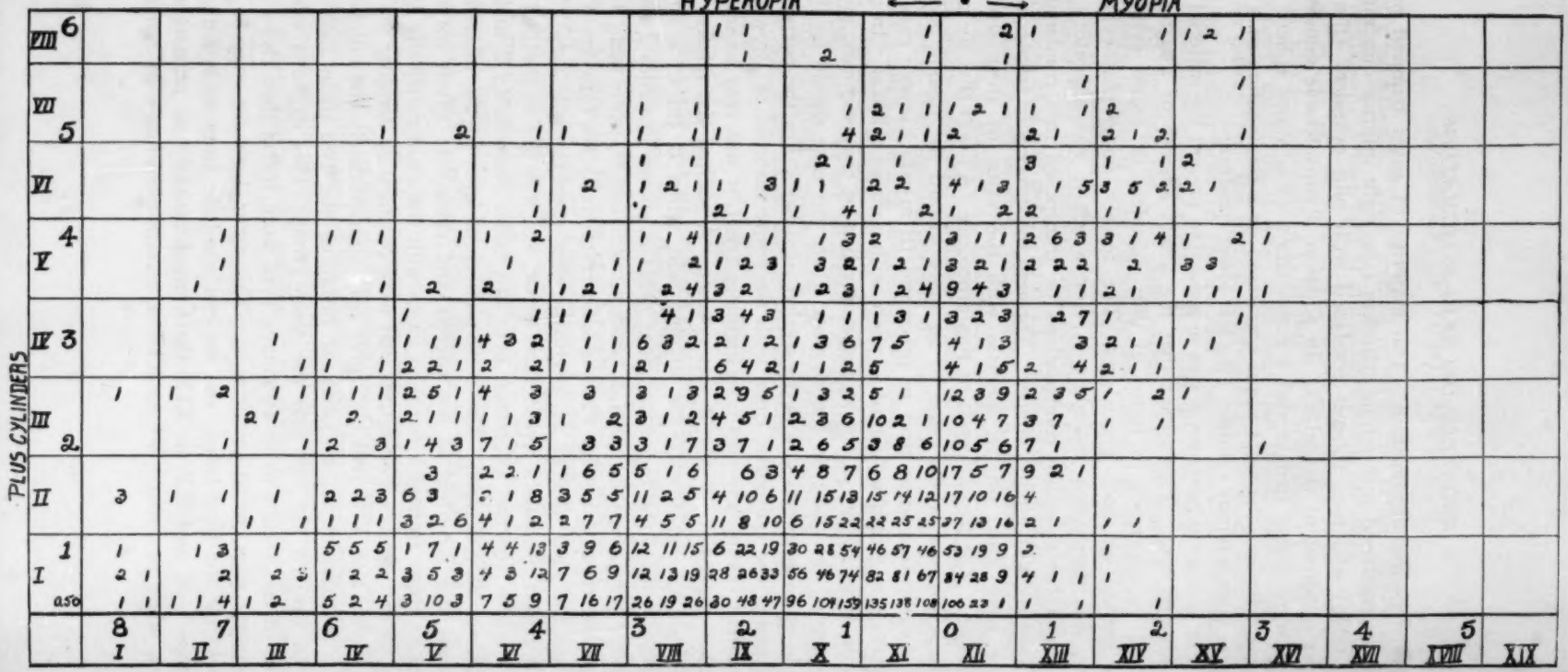

TABLE 1—A

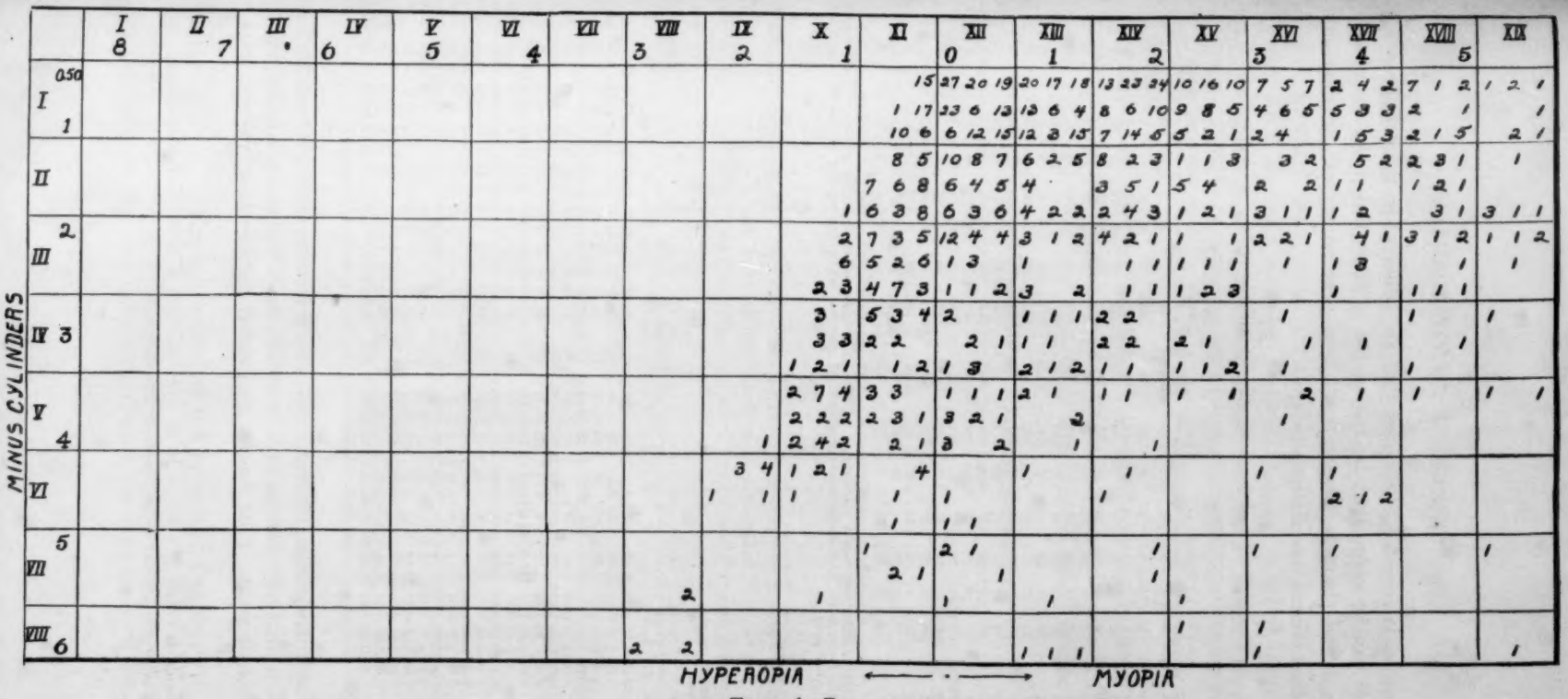

TABLE 1—B

* Each unit contains a number which is equivalent to the number of eyes found for each spherocylindric combination in the entire material. The arabic numbers represent diopters (spheres plus cylinders) while the Roman numerals indicate groups (0.75 diopters width) which will be carried throughout the paper.

matic eyes is found. The errors that are adherent to the procedures A and B can be eliminated by taking the arithmetical mean as a basis. Table 4 and chart 1 present $\frac{\text{A plus B}}{2}$ in percentages of the total sum of astigmatic and nonastigmatic eyes.

TABLE 2.—*Comparison of Results of Procedures A and B* *

A—Plus Cylinders								Total of		Total of	B—Minus Cylinders							
8	7	6	5	4	3	2	1	A	Spheres	B	1	2	3	4	5	6	7	8
0	0	0	0	0	1	3	6	10	I	40	9	2	10	11	6	5	3	3
0	0	0	3	0	4	2	12	21	II	79	18	12	22	10	7	6	2	2
0	0	0	0	2	6	3	9	20	III	96	23	20	19	13	12	7	1	1
0	1	0	4	2	10	10	31	58	IV	137	40	26	17	19	17	6	11	1
0	2	0	3	9	20	23	36	93	V	164	54	40	19	22	14	11	4	0
0	1	2	7	14	25	23	61	133	VI	202	81	41	42	17	14	7	0	0
0	1	3	6	7	12	41	80	150	VII	267	138	57	30	26	16	0	0	0
0	4	7	15	19	24	44	153	266	VIII	409	262	91	42	8	0	0	2	4
3	1	7	14	27	37	58	259	406	IX	732	551	145	26	0	1	9	0	0
2	5	10	15	16	30	101	644	822	X	958	818	80	13	13	27	5	1	0
2	8	8	14	12	36	137	760	977	XI	483	347	51	42	19	14	6	4	0
3	6	12	27	26	66	138	402	680	XII	317	193	55	38	9	14	3	5	0
1	6	11	19	18	45	73	135	308	XIII	251	154	64	12	10	6	1	1	3
1	7	14	15	26	52	55	104	274	XIV	229	114	71	26	10	3	2	2	1
4	2	15	39	18	21	31	117	247	XV	156	66	23	50	14	2	0	0	1
2	2	4	16	10	12	23	67	136	XVI	106	40	17	16	17	12	1	1	2
0	3	6	13	9	9	19	36	95	XVII	98	28	12	14	21	13	8	1	1
0	5	4	7	13	20	27	39	115	XVIII	84	21	13	10	6	19	13	2	0
4	1	3	6	8	12	18	8	60	XIX	53	8	7	5	2	17	10	3	1

* Each number represents the number of eyes in a single spherocylindric group. The Roman numerals from I to XIX designate the spherical groups, while the arabic numerals represent the cylindric groups.

TABLE 3.—*Figures in Table 2 Expressed in Percentage of Astigmatic Plus Non-Astigmatic Eyes for Procedures A and B*

Astigmatic Plus Non-astigmatic	A								Spherical	B							
Eyes	8	7	6	5	4	3	2	1	Group	1	2	3	4	5	6	7	8
21	0.00	0.00	0.00	0.00	0.00	4.76	14.28	28.58	I	42.84	9.52	47.6	52.36	28.56	23.80	14.28	14.28
50	0.00	0.00	0.00	3.00	0.00	4.00	2.00	12.00	II	36.00	24.00	44.00	20.00	14.00	12.00	4.00	4.00
53	0.00	0.00	0.00	0.00	3.78	11.32	5.66	17.20	III	43.38	37.72	35.84	24.52	22.64	13.20	1.88	1.88
95	0.00	1.06	0.00	4.24	2.12	10.58	10.58	32.80	IV	42.32	27.50	17.98	20.10	17.98	6.34	11.64	1.05
130	0.00	1.70	0.00	2.30	6.90	15.32	17.62	27.58	V	41.36	30.64	14.56	16.86	10.72	8.42	3.06	0.00
189	0.00	0.54	1.06	3.70	7.42	13.24	12.20	32.34	VI	42.94	21.74	22.26	9.02	7.42	3.72	0.00	0.00
259	0.00	0.40	1.16	2.32	2.70	4.64	15.82	30.88	VII	53.26	22.00	11.58	10.04	6.18	0.00	0.00	0.00
496	0.00	0.80	1.14	3.04	3.84	4.84	8.88	30.90	VIII	52.92	18.38	8.48	1.62	0.00	0.00	0.40	0.80
980	0.30	0.10	0.72	1.44	2.76	3.78	5.92	26.46	IX	56.20	14.80	2.66	0.00	0.10	0.92	0.00	0.00
1,800	0.12	0.28	0.56	0.84	0.90	1.66	5.62	35.90	X	45.80	4.48	0.72	0.72	1.52	0.28	0.05	0.00
1,426	0.14	0.56	0.56	0.98	0.84	2.52	9.62	53.36	XI	24.30	3.58	2.94	1.34	0.98	0.42	0.28	0.00
774	0.40	0.78	1.56	3.48	3.34	8.52	17.94	51.94	XII	24.94	7.10	4.90	1.16	1.80	0.38	0.64	0.00
362	0.28	1.66	3.04	5.24	4.96	12.42	20.14	37.26	XIII	41.28	17.16	3.22	2.68	1.60	0.26	0.26	0.80
297	0.34	2.36	4.74	5.08	8.78	17.58	18.60	35.16	XIV	38.76	24.14	8.84	3.40	1.02	0.68	0.68	0.34
171	2.32	1.16	8.76	22.80	10.52	12.26	18.10	68.32	XV	38.88	13.34	29.00	8.12	1.16	0.00	0.00	0.58
140	1.36	1.36	2.72	10.92	6.82	8.18	15.68	45.70	XVI	27.20	11.56	10.88	11.56	8.16	0.68	0.68	1.36
90	0.00	3.36	6.72	14.56	10.08	10.08	21.28	40.32	XVII	31.36	13.44	15.68	23.52	14.56	8.96	1.12	1.12
75	0.00	6.66	5.34	9.34	17.34	26.68	36.02	52.02	XVIII	28.14	17.42	13.40	8.04	25.46	17.42	2.68	0.00
49	8.16	2.04	6.12	12.24	16.32	24.48	36.72	16.32	XIX	16.32	14.28	10.20	4.08	34.68	20.40	6.12	1.02

The unavoidable incorrectness of these total sums led us to the decision that the comparison between the frequencies of astigmatic and nonastigmatic eyes was to be carried out by using the meridian [5]

5. It should be kept in mind that by "astigmatic meridian" is meant, that the meridian concerned was found in an eye combined with another meridian of different strength (minimum difference 0.5 diopter).

as the unit. In other words, the only correct way of giving the frequency of astigmatism was to group the meridians as follows: Among n meridians that were found to need a correction of, for instance, plus 4.00, there were, first, a meridians, which were combined with another main meridian differing from the former by 0.5 to 1.00 diopters (astigmatism group 1, as we expressed it, a^{I}), and, second, b meridians, which are combined with another meridian differing from the former by 1.25 to 1.75 diopters (astigmatism group 2, or as we expressed it, b^{II}), and so on.

After much thought, we have been unable to determine a better way of comparing frequencies of astigmatic and nonastigmatic eyes in which each individual unit was listed and counted in its proper place.

TABLE 4.—$\frac{A \text{ plus } B}{2}$ *Expressed in Percentage*

Spherical Group	1	2	3	4	5	6	7	8	Astigmatic Eyes	Nonastigmatic Eyes
I	35.70	11.90	26.18	26.18	14.28	11.90	7.14	7.14	140.42	—40.42
II	30.00	7.00	26.00	10.00	10.00	6.00	2.00	2.00	100.00	00.00
III	40.30	21.68	23.58	14.14	11.32	6.60	0.94	0.94	119.50	—19.50
IV	37.56	19.04	14.28	11.10	11.10	3.16	6.34	0.52	103.10	— 3.10
V	34.46	24.12	14.94	11.88	6.50	4.20	2.38	0.00	98.48	1.52
VI	37.64	16.96	17.74	8.22	5.56	2.38	0.28	0.00	88.78	11.22
VII	42.06	18.90	8.10	6.38	4.24	0.58	0.20	0.00	80.46	19.54
VIII	41.90	13.62	6.66	2.72	1.52	0.40	0.60	0.04	67.46	32.54
IX	41.32	10.36	3.22	1.38	0.76	0.82	0.50	0.14	58.50	41.50
X	40.80	5.05	1.18	0.80	1.18	0.42	0.42	0.06	49.90	50.10
XI	38.84	6.60	2.74	1.08	0.98	0.48	0.42	0.08	51.22	48.78
XII	38.44	12.52	6.70	2.24	2.64	0.96	0.70	0.20	64.40	35.60
XIII	39.26	18.64	7.82	3.82	3.42	0.65	0.90	0.54	75.12	24.88
XIV	36.96	21.36	13.20	6.08	2.34	2.70	1.52	0.34	84.50	15.50
XV	53.20	15.72	20.62	9.32	11.98	4.38	0.58	1.56	117.36	—17.36
XVI	36.44	13.62	9.52	9.18	9.54	1.70	1.02	1.36	82.38	17.62
XVII	35.84	17.36	12.88	16.80	14.56	7.84	2.24	0.56	108.08	— 8.08
XVIII	40.08	26.72	20.04	12.68	17.40	11.38	4.68	0.00	132.98	—32.98
XIX	16.28	25.50	17.24	10.20	23.46	13.26	4.08	5.10	115.12	—15.12

The results are given in table 5 and in chart 2. In this table, the respective frequencies for each group of astigmatism are expressed in percentages of the total number of meridians that fell into this group. In three groups, namely, I, IV and XV, there were more astigmatic meridians found than there were astigmatic plus nonastigmatic meridians. The difference, however, was almost negligible as the tabulation shows:

Group I excess 3.1 per cent equals 1.30 meridians
Group IV excess 4.94 per cent equals 9.34 meridians
Group XV excess 1.93 per cent equals 6.61 meridians

This excess was probably due to the fact that when the whole material of Brown and Kronfeld was reviewed the first time, a few cases were overlooked which were included when the material was gone over the second time.

In all the other groups the astigmatic meridians are less frequent than the total sum of astigmatic plus nonastigmatic eyes. The difference between the two figures represents the frequency of nonastig-

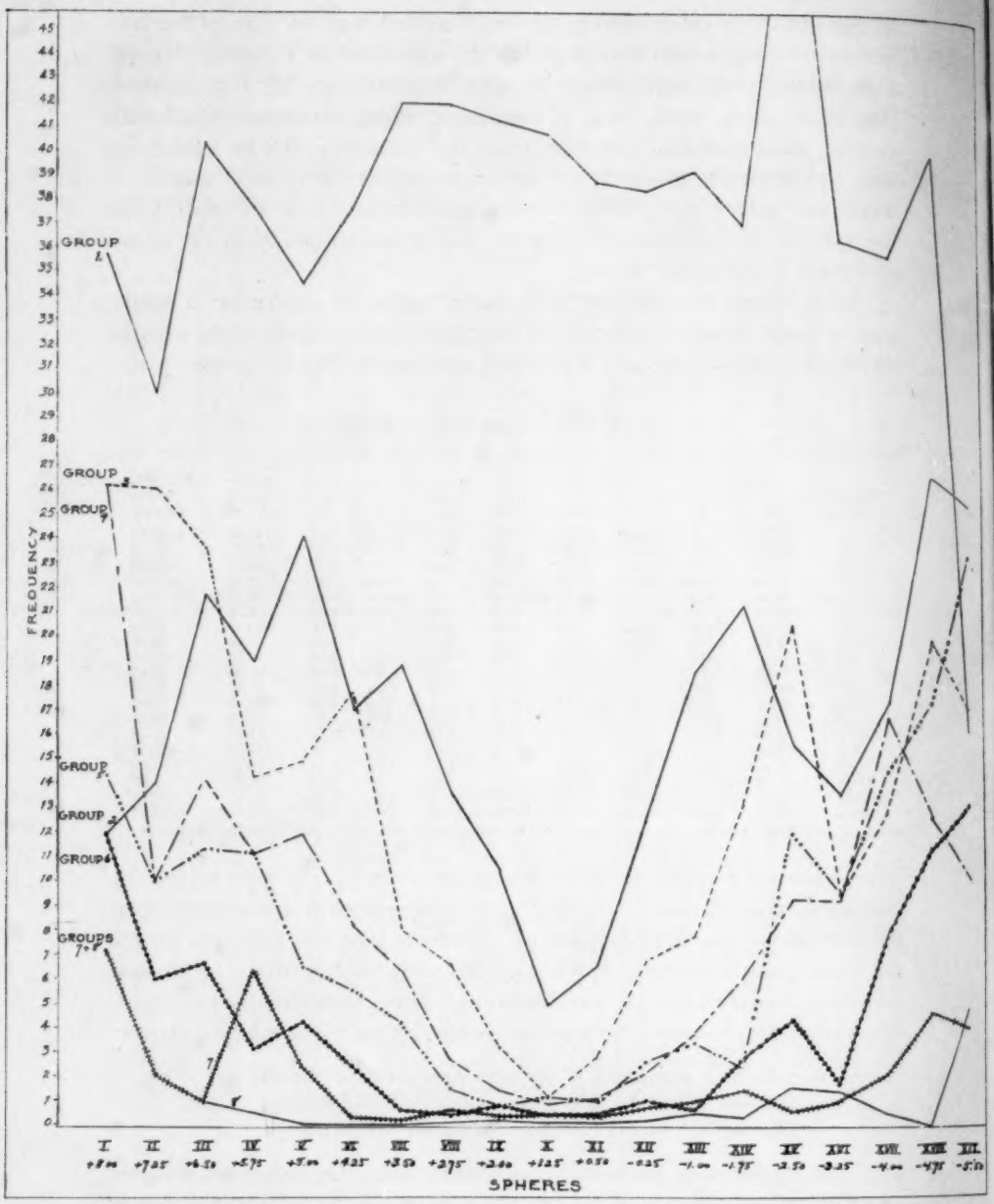

Chart 1.—Eight curves plotted from the figures in table 4. Group 1 represents astigmatisms of from 0.50 to 1.00 diopter; group 2, from 1.25 to 1.75 diopter; group 3, from 2.00 to 2.50 diopter; etc. The figures on the ordinate represent in percentage the ratio between astigmatic and the astigmatic plus nonastigmatic eyes for each spherical group.

matic meridians, or if they are divided by 2, the number of nonastigmatic eyes for each specific group.

From table 5 and chart 2 it can be seen that there is a marked difference in the frequency of an astigmatism of group 1 (0.5 up to 1.00) and an astigmatism of a higher degree. The latter becomes less frequent toward emmetropia, the former does not. The curve that represents group 1 is fairly regular. There is only one gross irregularity at the point that corresponds to a spherical refraction of minus 2.25, 2.50 and 2.75. This abrupt rise and fall is not beyond our expectations because of the relatively small number of cases investigated. By further studies we hope to increase our material and thus eliminate this irregularity. There is, however, the possibility that in this specific

TABLE 5.—*Relationship in Percentage Between Astigmatic Meridians and the Total Meridians (Astigmatic Plus Nonastigmatic) for Each Spherocylindric Group*

Spherical Groups	1	2	3	4	5	6	7	8	Astigmatic Meridians	Nonastigmatic Meridians
I	23.80	4.76	21.42	19.04	11.90	11.90	7.14	7.14	103.10	— 3.10
II	30.00	11.00	22.00	14.00	7.00	6.00	2.00	2.00	94.00	6.00
III	27.26	21.62	18.80	6.58	11.28	6.58	0.94	0.94	94.00	6.00
IV	37.63	19.08	13.78	14.84	9.01	3.18	5.30	2.12	104.94	— 4.94
V	34.20	23.94	14.82	11.40	5.32	4.18	2.66	0.76	97.28	2.72
VI	37.63	16.96	18.55	7.44	5.30	3.97	1.33	0.53	91.69	8.31
VII	42.07	18.91	8.11	6.37	6.37	1.93	0.97	0.77	85.50	14.50
VIII	41.92	13.64	6.67	3.74	3.74	2.22	0.81	0.40	73.14	26.86
IX	41.31	11.93	3.67	2.40	1.48	0.97	0.10	0.15	62.01	37.99
X	41.02	6.75	2.13	1.12	1.48	0.42	0.17	0.06	53.15	46.85
XI	41.93	7.46	2.84	1.58	1.05	0.49	0.42	0.07	55.84	44.16
XII	35.88	12.68	6.11	2.27	2.80	0.98	1.04	0.13	61.89	38.11
XIII	33.67	13.52	7.87	3.31	3.45	1.79	0.55	0.14	64.30	35.70
XIV	35.15	14.53	10.65	5.75	3.55	2.70	0.85	0.17	73.35	26.65
XV	53.45	14.31	9.34	7.30	11.68	4.09	0.59	1.17	101.93	— 1.93
XVI	36.49	12.62	6.82	3.75	5.46	1.36	0.68	1.36	68.54	31.46
XVII	35.78	16.21	7.83	5.03	7.27	2.80	1.12	0.00	76.04	23.96
XVIII	36.02	10.01	6.00	6.67	4.00	3.34	3.34	0.00	69.38	30.62
XIX	16.32	15.30	8.16	5.10	3.06	0.00	2.04	0.00	50.52	49.48

refractive group (minus 2.25 to 2.75) a small amount of astigmatism is more frequent than in any other group, because this group represents mostly cases of so-called school myopia, or, as we might call it, "growth myopia," in which unbalanced states may be more common.

In general, the curve of group 1 remains on a fairly constant level between the refractions of plus 3.50 and minus 5.00, while it drops suddenly on the myopic end and gradually on the hyperopic end. Between the limits mentioned, the frequency of an astigmatism of group 1 is constant and therefore parallels the general refraction curve which represents the astigmatic plus the nonastigmatic meridians. This fact speaks in favor of the differentiation of a "physiologic" from a "pathologic" astigmatism, as has been done in ophthalmology for a long time. Donders thought that 1 diopter would be the proper upper limit for the physiologic astigmatism. Our curve indicates that this limit is about right, because an astigmatism of 1 diopter belongs to the first group, while one of 1.25 diopters falls into group 2.

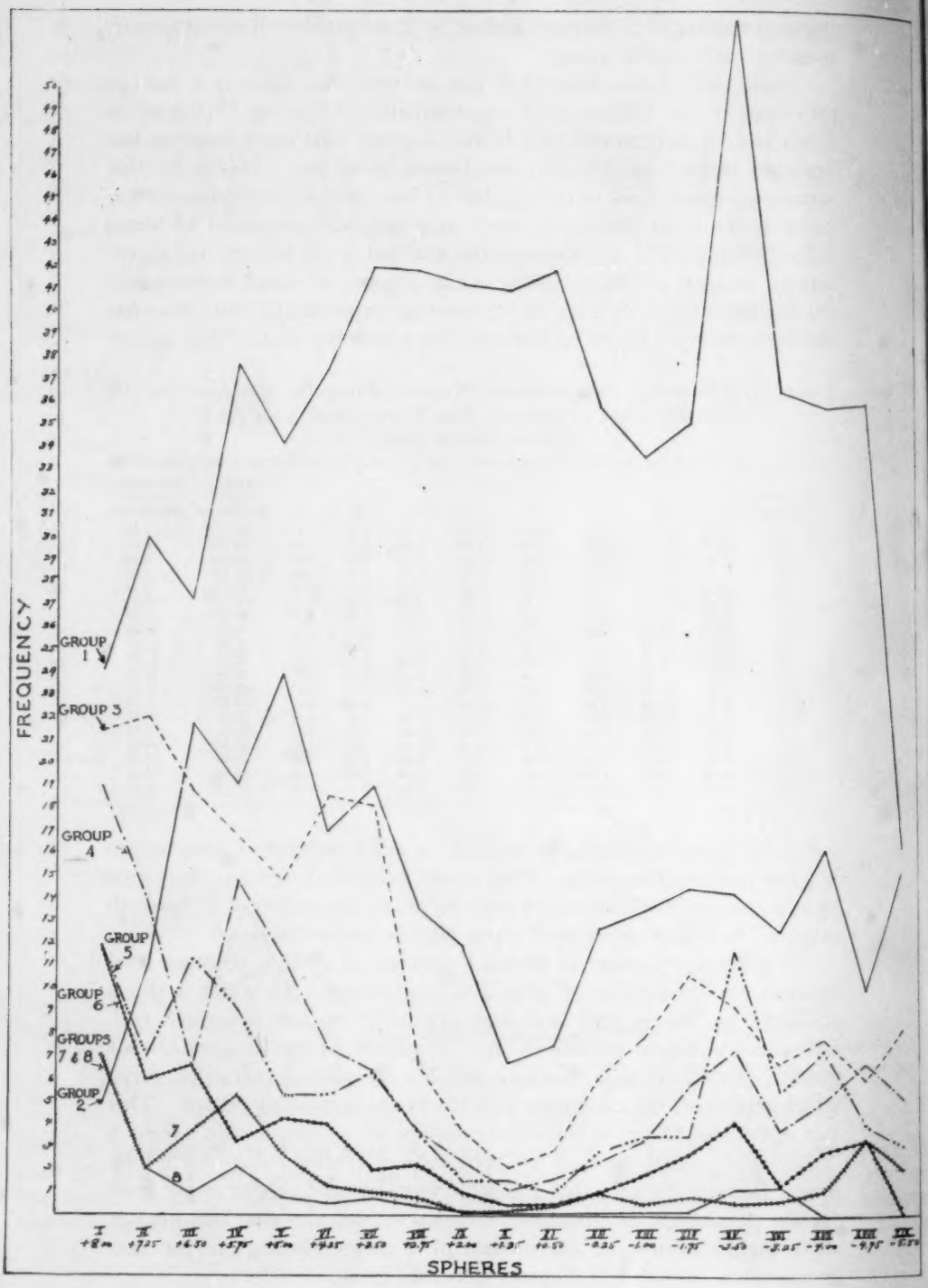

Chart 2.—Eight curves representing the frequencies of astigmatism plotted from the figures in table 5.

For the higher degrees of astigmatism (over 1.00 diopter) it can be seen that their minimum frequency lies at plus 1.25, which is the most common refraction according to Brown and Kronfeld. This point, which we might call the center of gravity in refraction, is therefore defined as the point that represents the maximum frequency of occurrence, and—at the same time—the minimum frequency of higher astigmatism. From this center of gravity, the curves for the higher degrees of astigmatism rise toward their terminations but to different levels. The slight asymmetry of each curve is produced by the higher frequencies throughout the hyperopic side and the lower frequencies throughout the myopic side. From this we can conclude that a hyperopic eye of a certain degree is more apt to have a higher grade of astigmatism than a myopic eye of the corresponding degree. These degrees can be found by considering plus 1.25 as the center around which the other refractive states should be placed, as follows:

$$+1.25 \begin{matrix} +0.5 & -0.25 & -1.00 & -1.75 & -2.50 & -3.25 & -4.00 & -4.75 & -5.50 \\ +2.00 & +2.75 & +3.50 & +4.25 & +5.00 & +5.75 & +6.50 & +7.25 & +8.00 \end{matrix}$$

From the biologic point of view, the distribution of astigmatism over the various refractive states shows that Steiger's theory, in which every refractive state is just a normal variation of normal refraction, must not be carried too far. There are at least two points in which so-called emmetropia (in our opinion plus 1.25) dominates all the other refractive states, and these are (1) its greater frequency of occurrence and (2) its very strong tendency to form nonastigmatic eyes. The more the refractive state differs from emmetropia, the more likely is the occurrence of an astigmatic eye. The relatively greater frequency of astigmatism on the hyperopic side may be explained by the fact that the hyperopic eye with its shorter anlage is more anomalous than the myopic eye with its longer anlage.

A few words must be said about the age of our patients. When dealing with refraction in general, a person over 25 is usually considered an adult representing the final stage of the normal development of the eyeball. This does not hold true for the astigmatism. Since the introduction of the ophthalmometer, the living changes of the corneal astigmatism have become well known. In the literature we also find a few reports on changes of the total astigmatism (Pomeroy,[6] Theobald[7] and Jackson[8]); yet we do not know how frequent this phenomenon actually is. For the time being, it can be assumed that these changes are slight and scarcely noticeable statistically for the higher degrees of astigmatism, but for the physiologic astigmatism they are

6. Pomeroy, O.: Progressive Hyperopic Astigmatism, Tr. Am. Ophth. Soc., 1867, p. 30.

7. Theobald, S.: Progressive Astigmatism, Tr. Am. Ophth. Soc., 1885, p. 29.

8. Jackson, E.: Progressive Hyperopic Astigmatism, Tr. Am. Ophth. Soc., 1890, p. 676.

marked. It means that a small amount of astigmatism with the rule that is found at the age of 30 is likely to be an astigmatism against the rule at the age of 60. Numerous statistics (Hess [9]) have been published on this subject. Our material has shown the same (table 6). Statistically, this means that at the age of 50, for instance, a considerable number of patients who had slight astigmatism with the rule at the age of 30 show no astigmatism at all. If one were really interested in finding the actual frequencies of low degree of astigmatism, it would be necessary to confine one's statements to periods of five or ten years, which would require an immense quantity of material. Therefore, the figures that we have given for the frequencies of physiologic astigmatism are not absolutely correct, because they include people of every age. This also applies to the main point of this paper, the frequency of astigmatism over 1.00 diopter. Before the changes of total astigmatism during life could be studied statistically, it was necessary to show on a large material how the distribution of astigmatism takes place irrespective of the age of the bearers.

TABLE 6.—*The Frequency of Astigmatism With and Against the Rule*

Astigmatism in Diopters	Age					
	0 to 30		30 to 40		40 to 60	
	With	Against	With	Against	With	Against
0.50-1.00	32.5%	17.5%	74.5%	25.5%	68%	32%
1.25-1.75	94.5%	5.5%	79 %	21 %	69%	31%

SUMMARY

The frequency of astigmatism was studied by using refractions that were made under complete atropine cycloplegia. The material investigated is that which was used by Brown and Kronfeld for their "Refraction Curve in the United States" (approximately 10,000 eyes). It has demonstrated that the differentiation between physiologic and pathologic astigmatism is justified, and that the curve for the physiologic astigmatism parallels the general refraction curve. The curves for the higher degrees of astigmatism (over 1.00 diopter) show their lowest points at the refraction of plus 1.25, from which point they rise toward the extremes of hyperopia and myopia, reaching slightly higher frequencies on the hyperopic than on the myopic side. The refraction of plus 1.25 has been shown to be the most frequent one by Brown and Kronfeld, and as a result of this investigation the same refraction is found to be the one with the strongest tendency to form nonastigmatic eyes.

9. Hess, C.: The Anomalies of Refraction and Accommodation of the Eye in Graefe-Saemisch: Handbuch der Gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1901-1911, vol. 8, part 2, p. 420.

THE CENTRAL NERVOUS SYSTEM CONTROL OF THE OCULAR MOVEMENTS AND THE DISTURBANCES OF THIS MECHANISM

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(Concluded from p. 661)

DISTURBANCES OF OCULAR MOVEMENT CAUSED BY INTERFERENCE WITH THE NUCLEI AND INTRA-AXIAL COURSE OF THE OCULOMOTOR NERVES

The disturbances of eye movement dependent on interference with the nuclei or the intra-axial course of the oculomotor nerves present similar evidence to that discoverable in lesions affecting the extraneural course of the oculomotor nerves. The results of these lesions may be of all kinds and combinations. The nuclei may be completely or incompletely involved, either bilaterally or unilaterally, and any one or more or all of the six oculomotor nuclei may be implicated.

The disturbances produced by lesions of the root fibers within the brain stem are similar to those caused by lesions affecting the nuclei. On account of the divergence of the root fibers from each other, the evidence of intra-axial involvement is much less apt to be bilateral, and such lesions are more apt to be limited to a more or less complete paralysis of the muscles specifically innervated by the nerve concerned.

Wilbrand and Saenger established the six following criteria for differentiating between nuclear, root and gaze paralysis:

1. In unilateral lesions of the cortex and of the supranuclear pathways or the association centers, one or more muscles of both eyes are affected, whereas in nuclear palsy the mobility of only the affected muscle is involved.

2. In the lesions that involve the specific center, single movements only are affected, while the same eye muscles for other movements are intact. In the nuclear and root palsies, all forms of movement of the affected muscles are disturbed.

3. In pure gaze paralysis, there is no double vision, since the eyes remain parallel.

4. In supranuclear palsies, the eyes are still subject to the reflex control of the vestibular apparatus. In nuclear or center paralysis, this is affected also.

5. An isolated, unilateral nuclear palsy of the abducens nerve may be confused with palsy of lateral gaze, but the retention of the internal

movement of the contralateral eye will distinguish between the two disturbances.

6. A bilateral, isolated palsy of the internal rectus can be differentiated from a paralysis of the movements of convergence by the failure in the former case of all movements of the internal rectus muscle.

Involvement of the intra-axial course of the emergent fibers can usually be recognized by the discovery of other evidences of involvement of the brain stem itself, either as associated nuclear palsies affecting the facial or the trigeminal nerve or by involvement of the long fiber tracts that pass upward and downward within the substance of the neuraxis conveying sensory impressions upward or transmitting motor stimuli downward to the limbs.

An isolated lesion of the abducens or trochlear nerve usually means a root lesion if it is unaccompanied by other symptoms of neurologic involvement. The association of paralysis of the abducens nerve with the facial nerve is frequent where either the nucleus or the root fibers may be involved. The total picture of the neurologic disease is important in the differential diagnosis of oculomotor palsies.

The various diseases of the nuclei, neuraxial centers or the root fibers within the neuraxis may be grouped as follows:

Congenital Aplasia of the Oculomotor Nuclei.—This condition may be found to affect all or any one of the oculomotor nuclei, and seems to depend on a failure of differentiation of the cells that are destined to give rise to the oculomotor nuclei. This is essentially a dysplasia or aplasia of the neurogenic elements.

Chronic Progressive External Ophthalmoplegia.—Recent work reported by Langdon and Cadwalader [6] in 1928 would seem to indicate that this condition, which has been considered as due to an idiopathic degeneration of the muscles themselves, may be due to a degeneration of the oculomotor nuclei unassociated with other evidence of involvement of the brain stem. This may perhaps fall within the group of the abiotrophies by which the actual viabilty of the cells may be affected, producing an early onset of disturbance in oculomotor function after a period of perfect function. This may progress for a period of from thirty to forty years, may cease at any time or may produce a total external ophthalmoplegia. It is usually ushered in by a ptosis and progresses symmetrically, affecting one after another of the oculomotor nuclei. On account of the symmetricality of the involvement, there is usually no diplopia. According to the claim of Langdon and Cadwalader, their case is the only one that has come to autopsy, and in their examination they found that the cells of the oculomotor nuclei, although fairly well stained, showed a slight chromatolysis. The cells were slightly diminished in number, and there were some variations in

the size and shape of the cells. The axis cylinders were apparently normal. The nuclei of Darkschewitsch and of Perlia together with the Edinger-Westphal nucleus were normal, as were also the fibers connecting the various parts of the nuclei. The nerve roots seemed smaller but were well stained. These observations are similar to those described by Greenfield and by Stern [15] and others in the Werdnig-Hoffmann type of progressive spinal atrophy of childhood, which is considered to be a chronic neuronal degeneration, thus bringing these two conditions into line with each other.

Intoxication.—The activity of the oculomotor nuclei may be directly affected by the various intoxications to which the central nervous system is subject. These may be of the exogenous type, resulting from exposure to the heavy metals or noxious gases, to the ingestion of liquids such as alcohol or to other drug or food poisoning. Polioencephalitis superior of Wernicke is described as almost regularly due to the effects of alcohol.

The endogenous intoxications are those so familiarly known, and are usually produced by nephritis, diabetes, gout, exophthalmic goiter and other disturbances of metabolism.

The Infections of the Nervous System.—The infectious diseases of the nervous system are apt to produce involvement of the oculomotor nuclei. These may be polioencephalitic, similar to poliomyelitis, in which the disease affects these particular nerve cells, and in this case is called polioencephalitis superior. Similar disturbances also appear in chronic polioencephalitis, which is essentially a slow progressive degeneration of the nuclei of the spinal cord and of the brain stem. The oculomotor nuclei are frequently involved in the other acute infections of the nervous system, especially in epidemic encephalitis. The more chronic types of inflammation, such as those produced by tuberculosis in the form of a tuberculoma or by syphilis, may frequently affect the oculomotor nuclei. Syphilis causes a great number of the oculomotor paralyses and may produce disturbances by attacking the nuclei, the connecting fiber tracts or the interaxial course of the root fibers.

Involvement of the nuclei or root fibers is also seen in many of the acute infectious diseases, such as typhoid, influenza, erysipelas, pneumonia, rheumatic fever or the exanthems. Diphtheria is a frequent source of oculomotor palsies, and may attack the nuclei or the efferent fibers as a true postdiphtheritic neuritis. Abscesses may also cause isolated or associated oculomotor palsies.

Neoplasms.—Neoplasms of the brain stem often affect the nuclei or fibers connecting the oculomotor nuclei or forming the emergent root.

15. Greenfield, J. G., and Stern, R. O.: Anatomical Identity of Werdnig-Hoffmann and Oppenheim Forms of Infantile Muscular Atrophy, Brain **50**:652, 1927.

The course of the disease and the involvement of other adjacent structures provide the criteria by which the differential diagnosis must be made.

Vascular Accidents.—Disease of the blood vessels in the form of hemorrhage, thrombosis or embolism may affect the small terminal arteries that supply the oculomotor nuclei. In these cases, the association of other symptoms together with the type of onset and collateral evidence supplied by the condition of the blood vessels wherever they may be seen or felt, together with the estimation of the blood pressure, provide the differential diagnostic criteria.

Injuries.—Injuries to the central nervous system, while they do not affect the cranial nerve nuclei or their fibers so extensively as they do the extra-axial course of the fibers, are a frequent cause of oculomotor palsies of one type or another. Trauma causes hemorrhages which may affect the nuclei or the emergent fibers.

Other Causes.—Myasthenia gravis, in which lymphorrhages have been found not only in the muscles affected but also within the central nervous system itself, may involve the nuclei. The degenerative diseases of the nervous system, such as multiple sclerosis, combined sclerosis, Friedreich's ataxia and others, may produce oculomotor disturbances. Exophthalmic goiter may be associated with oculomotor palsies either as a result of an endogenous toxemia or as a mechanical stretching of the muscles of the eyes. Oculomotor palsies may also appear as recurrent paroxysmal disturbances in migraine that may affect the isolated oculomotor nuclei or produce gaze disturbances. Periodic oculomotor palsy has also been observed without migraine.

Paralysis of the abducens nerve itself as a single symptom may often be associated with an internal hydrocephalus or with a diffuse increase in intracranial pressure. Under such circumstances the abducens palsy has no direct localizing value, as it may be due to pressure on the nucleus or to a distortion of the extra-axial course of the nerve from either dislocation or pressure.

The infrequency of bilateral abducens palsy is in contrast with the marked frequency of bilateral paralysis of the oculomotor nerve due to neoplasms or inflammatory processes arising along the base of the brain. Bilateral involvement of the oculomotor nuclei may also appear in extensive pseudobulbar palsy.

Isolated unilateral paralysis of the trochlear nerve is rare but has been described and must be looked for. It may result from any of the aforementioned causes. Lesions in the superior medullary velum may result in a bilateral involvement of the two nerves at their decussation.

OCULOMOTOR SYNDROMES

There are a number of syndromes that involve the oculomotor nuclei and are sufficiently well recognized to be worthy of description.

The Syndrome of Superior Alternating Hemiplegia.—This syndrome is also called Weber's syndrome, and presents a complete external and internal ophthalmoplegia on the same side as the lesion, with a crossed hemiplegia of the upper motor neuron type involving the upper and lower extremities of the opposite side either partially or completely. With lesions properly placed this may be a tetraplegia with a bilateral involvement of the oculomotor nerves. There are a number of combinations of Weber's syndrome, including variations of the unilaterality or bilaterality of the ophthalmoplegia with hemiplegia or tetraplegia. The syndrome may be associated with evidence of involvement of the tegmentum of the midbrain and the red nucleus, with tremors, hemianesthesias, etc.

The entire syndrome may not appear at one time, but any portion may appear first, to be followed later by the development of the complete syndrome.

The Tegmental Syndrome of the Midbrain.—This syndrome is called the syndrome of Benedict, and is characterized by an involvement of the intra-axial course of the third cerebral nerve together with involvement of the red nucleus, resulting in choreiform tremors, a hemiasynergia and a hemichorea of the opposite upper and lower extremities.

The Syndrome of Middle Alternating Hemiplegia.—This syndrome, also called the syndrome of Millard-Gubler, affects the abducens nerve of one side together with the pyramidal tract destined to the control of the upper and lower limbs of the opposite side. Often it is the result of an inflammation of the meninges immediately overlying the lower border of the pons varolii or of tumors or other lesions in this vicinity, affecting the abducens nerve close to or at its exit from the brain stem. This lesion is also found to be frequently associated with other evidences of neuraxial involvement. At times the facial nerve is paralyzed on the same side. It infrequently is associated with herpes zoster ophthalmica. The association of these symptoms is sufficient to determine the focal character of the lesion. There may also be other evidences of involvement of the brain stem, such as a hemianesthesia of the side opposite to the lesion, affecting the discriminative or affective types of sensibility or both.

The Syndrome of Foville.—This syndrome is characterized by a loss of lateral conjugated gaze, the external rectus muscle of one eye and the internal rectus muscle of the other eye being involved. It may be bilateral. It probably results from an involvement of the center for

lateral gaze situated near the abducens nucleus. There may also be evidence of more extensive involvement of the metencephalic tegmentum.

Guillain and his co-workers [16] described the onset of paralysis of lateral gaze to the left, which was followed by evidence of involvement of the structures of the tegmentum of the pons varolii. This was followed by a paralysis of gaze to the right and some involvement of convergence movement. The movements of upward and downward gaze were fully retained.

Dupuy-Dutemps, Laignel-Lavastine and Desoille [17] described a case of ophthalmoplegia beginning with an isolated palsy of the left external rectus muscle and resulting in an internal strabismus with diplopia. With this was associated a left facial paralysis. During the course of two months, this condition extended in such a way as to involve the lateral movements of both eyes equally, producing a complete paralysis of lateral movement.

HYPERKINETIC DISTURBANCES

This type of disturbance is much less common than is the hypokinetic or paralytic type of oculomotor dysfunction. Cramps and isolated movements of the eyeballs, however, have been described. The presence of single muscle spasms is rarely seen, but I observed this recently in a case of multiple sclerosis in which in between the nystagmoid movements, which were present even on forward fixation, isolated rapid twitches of the external rectus muscle producing an outward deviation of one eye alone were observed.

Brownell [18] described a primary spasm of the superior oblique muscle which in his patient produced a sense of dizziness and diplopia on looking downward. The patient also suffered from considerable inability to judge distances.

These cramps often affect the muscles supplied by the oculomotor nucleus, particularly the levator palpebrae superioris, the internal rectus and the internal muscles of the eyeball. They may be the result of irritation of the nuclei or emergent fibers of the nerves within the brain stem from some cause such as intoxication or infection. Eye muscle cramps from general diseases of the nervous system, such as chorea, athetosis, tetany, Thomsen's disease, syphilis, convulsive disorders and migraine, have been reported.

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16. Guillain, G.; Thevenard, A., and Thurel, R.: Syndrome de la Calotte protubérantielle caracterisé par double syndrome de Foville, origine infectieuse probable, Rev. neurol. **1**:654, 1927.

17. Dupuy-Dutemps; Laignel-Lavastine, and Desoille, H.: Un cas de paralysie complète dans les deux sens des mouvements de latéralité des yeux, Bull. et mém. Soc. méd. d. hôp. de Paris **52**:1527, 1928.

18. Brownell, M. E.: Primary Spasm of the Superior Oblique Muscle, Am. J. Ophth. **11**:41, 1928.

The lesions that affect the pathway by which the various oculomotor nuclei are linked together in harmonious action are caused by the various pathologic processes that have already been named, and that in this case affect the posterior longitudinal fasciculus. Involvement of this bundle serves to break up the internuclear linking of the various oculomotor nuclei in the production of conjugated movement. In gaze movement one eye may execute the wishes of the person, whereas the normally associated movement of the other eye does not take place. This is particularly seen in the disturbances of lateral gaze, in which the external rectus is active but the associated action of the internal rectus muscle of the other eye does not take place.

Antoni [19] described a case in which the internuclear connections over the posterior longitudinal fasciculus were broken by a lesion. In this instance, the eyes when turned to one side showed a failure of adduction of the contralateral eye with resulting divergent strabismus and diplopia with nystagmus of considerable amplitude. This indicated a paralysis of the internal rectus. There was, however, a maintenance of convergence in which both internal recti muscles acted normally. This sort of disturbance is clearly the result of an interference between the center for lateral gaze and the internal rectus segment of the opposite third nerve nucleus. The presence of a retained convergence reaction and the fact that vestibular irritation failed to produce a nystagmus in the involved eye indicated that the lesion was situated in the connecting fibers between the nuclei. Antoni found this situation in three patients, all of whom were suffering from multiple sclerosis.

In connection with the reflex disturbances of the eye, the most important is nystagmus. This whole subject is so extensive and complex that it is impossible to enter into all of the various factors that are operative in its production.

The movements of nystagmus show a definite pattern, and are usually constant when sought for by various means. They possess a rhythmic character and may be associated with other evidence of involvement of the central nervous system.

Nystagmoid movements are occasional, irregular, oscillatory movements that may be seen on movement of the eyes in any direction. They are inconstant and irregular and have no definite pattern.

Oscillatory Nystagmus.—In this type the movements are relatively equal, and the movements in both directions are rapid and regular.

Rhythmic Nystagmus.—Rhythmic nystagmus presents a slow and a rapid component which affects both eyes equally. Such a component can

19. Antoni, N.: Eine eigenartige symmetrische Motilitätstörung der Augen Syndrome der internuclearen Langsbündels, Jahrb. f. Psychiat. u. Neurol. **45**:15, 1927.

be brought out by directing the gaze in one or another direction, and may be lateral or vertical or may possess a rotary component. This type of nystagmus is usually acquired and disturbs the patient, since it interferes with vision. The usual cause is believed to be an irritation of the vestibular apparatus, the activity of the vestibule producing the slow deviation of the eyes away from the point of fixation, the rapid component being supplied by the higher centers in bringing the eyes back to the point of fixation.

Fixation Nystagmus.—Fixation nystagmus is usually found in multiple sclerosis, and may be explained on the same basis as the intention tremor that is so characteristic of this disease. It is usually found in lateral gaze, but it may be found in vertical, diagonal or mixed deviations of the eyes.

Unilateral Nystagmus.—This type of nystagmus has been described, but it is probably the result of the breaking up of the linking between the two eyes due to the progress of the disease producing the nystagmus, so that the stimuli caused by the irritation are blocked in their passage to one or the other eye.

Miners' Nystagmus.—Miners' nystagmus is usually attributed to the insufficient light in which miners work and to the unusual strain on the eye muscles that is occasioned by their habitual posture in carrying on their work.

Hereditary, Familial or Congenital Nystagmus.—These disturbances in the eye movements are occasionally seen and probably depend on congenital dysplasia, by which the proper control of the ocular movements and their response to reflex stimuli is not perfectly acquired. Often other congenital abnormalities of the central nervous system or of the systemic structures are also present. The nystagmus is usually continuous, does not seem to interfere with vision and occasions no distress.

Nystagmus is also seen in a number of miscellaneous conditions. It is found in weakness of the ocular muscles, in albinism and in conditions that diminish the acuity of central vision, and it is seen in degenerative conditions such as those associated with long-continued convulsive states, color blindness, retinitis pigmentosa, etc. Nystagmus is also seen in rapidly developing cerebral anemia or hyperemia, with fever and with intoxication with barbital, sulphonethylmethane, hydrocyanic acid, mercury, formic acid, antipyrine, sodium arsinate, arsenic, quinine, potassium iodide, iodoform, salicylic acid, carbon dioxide, chloroform, codeine, cocaine, alcohol, nicotine, lead and food poisoning.

Nystagmus is also often found in association with other evidences of the tremor-producing tendency, particularly with movements of the head. Nystagmus has been described from lesions in the cerebral hemi-

spheres, but the authenticity of this cause for nystagmus is open to question, and it may be the result of multiple lesions within the nervous system. Investigation of cerebellar tumors by Mills, Frazier, Weisenburg and others has shown that nystagmus is a most frequent symptom. It is particularly common in cerebellopontile angle tumors. It is questionable whether nystagmus ever is the result of isolated cerebellar lesions. In all probability, when it appears in such conditions it is associated with vestibular involvement, by pressure on the root of the vestibular nerve, on the reception nuclei of the vestibular complex or on the roof nuclei of the cerebellum.

It is occasionally seen in diseases of the spinal cord, particularly of the upper part of the cervical cord, and when present under these circumstances it is explained by a retrograde irritation of the descending Deitero-spinal fibers that pass down into the spinal cord. It is occasionally seen in tabes dorsalis, Friedreich's ataxia, syringomyelia and syringobulbia. It is present also in a number of other conditions affecting the nervous system such as paralysis agitans, myasthenia pseudoparalytica, spasmus nutans, turmschadel, myoclonus, amaurotic family idiocy, acromegaly, and with intestinal parasites.

THE CONTROL OF OCULAR MOVEMENTS BY THE EXTRAPYRAMIDAL SYSTEM

In discussing the extrapyramidal control of the ocular movements, one is venturing on a field that is not only difficult on account of its great complexity, but also uncertain on account of the indefiniteness of our information in regard to many of the elements that are concerned in the extrapyramidal control of voluntary movement. According to the current conception of the extrapyramidal system, it is composed of two separate components. The first component is supplied by the cerebellum, which is generally conceded to provide the muscles of the extremities and of the trunk and of the face with the quality of synergic action by which muscle groups are brought into a condition of harmonious interplay with one another for the purpose of producing smooth, accurate and minutely modulated movements. This control is exerted over groups of muscles which, although antagonistically placed anatomically, as flexors and extensors, abductors and adductors, external and internal rotators, are physiologically synergists acting together with the most accurately graded changes in tone in order to complement and regulate each others' actions. This result is brought about by two factors, a dominant and a check element in all movements, by which the major incident of the voluntary effort falls on the dominant element in the movement until the object of the movement is about to be attained, at which time the check element brings the movement to a finely graduated termination.

This conception of cerebellar function has been promulgated by Mills and Weisenburg [20] and by Tilney and Pike.[2] As already mentioned, it is in conflict with the views of Sherrington, who believes that the muscles act antagonistically and are reciprocally innervated, and that inhibition is an active factor in the suprasegmental control of muscle movement. Whether this synergic activity of the cerebellum is exerted also over the ocular muscles is a question for debate. It is certain that cerebellar lesions do not often produce an asynergia of the ocular movements, and operative removal of the cerebellum does not seem to bring about any definite incoordination in ocular activity. It would seem to be true that the great majority of proved superficial cerebellar lesions seen at the Neurological Institute, particularly in connection with neoplasms of the cerebellum, do not regularly show a nystagmus or any asynergic involvement of the ocular muscles.

Orzechowski [21] described a dysmetric ataxia of the eyes which he believed was due to a cerebellar lesion. In this condition he described certain ocular hyperkineses that are characterized by a variable chaotic agitation of the eyeballs. This, he stated, is of such a character as to defy analysis, and he termed it dysmetric ataxia. The movements may be single or multiple and are of unequal amplitude. They seem to disappear on extreme lateral deviation, contrary to the usual manifestations of nystagmus. He stated that this disappearance apparently is due to mechanical factors, such as interference by the edge of the orbit with the ocular movement and the nonextensibility of the antagonistic musculature. He described it as a cerebellar dysmetria, and stated that other associated cerebellar signs were found in connection wtih this oculomotor disturbance. The two eyes are equally affected.

Russell, working under the direction of Sherrington, was able to show that operative lesions on the cerebellum and the peduncles of the cerebellum produce deviations of the opposite eye. This they explained as being due to a disturbance in the tonic control which the cerebellum is supposed to participate in, in connection with other tone-controlling centers.

The consensus would seem to indicate that there is no direct synergic control of the eye muscles by the cerebellum.

The other component of extrapyramidal control of the oculomotor muscles is supplied by the so-called striatal system. This system is controlled by the basal ganglions and associated structures, including the caudate nucleus, the putamen, the globus pallidus and the smaller masses of nuclear material, the corpus subthalamicum of Luys, the

20. Mills, C. K., and Weisenburg, T. H.: Cerebellar Symptoms and Cerebellar Localization, J. A. M. A. **63**:1813 (Nov. 21) 1914.

21. Orzechowski, C.: De l'ataxie dysmetrique des yeux, Jahrb. f. Psychol. u. Neurol. **35**:1, 1927.

substantia nigra and the nucleus ruber. The striatal component of extrapyramidal control is supposed to contribute the grace, fluidity, automaticity and rhythm which are so characteristic of normally executed movements of precision by the musculature of the human body. It is through the study of patients who have lost this contribution to motor organization that the importance of its presence in animal movement becomes apparent. This is particularly shown in those persons who have, by reason of birth accident, agenesis, inflammation or other cause, suffered a loss of the supervision of the cortex over the action of the corpus striatum and its associated structures, or who by reason of disease in the corpus striatum manifest hyperkinetic phenomena that may be due to either the loss of cortical inhibition or the actual stimulation of the gray matter of the basal ganglions. These conditions are seen particularly in chorea, bilateral athetosis, Wilson's disease, progressive lenticular degeneration, paralysis agitans and the familiar postencephalitic sequelae, particularly those characterized by tremor and rigidity. The chief characteristics evidenced by disease of the system controlling the extrapyramidal tract are abnormal involuntary movements, tremor and hypertonus. Usually all three of these components are equally mixed in the production of the particular syndrome, and as yet no clear-cut differentiation between the contributions of the various parts of the extrapyramidal system and these symptoms has been made. It is therefore a matter of considerable difficulty to make any definite statement as to the effect of disease of the extrapyramidal system on the oculomotor mechanism. The results as seen, however, would show that from an interference with this extrapyramidal system certain abnormal involuntary movements of the eyes make themselves manifest. These may be in the form of quick, irregular jerking movements similar to those seen in chorea. In addition, athetotic movements of the eyeballs may appear in disease of the basal ganglions. There is also apparent, particularly in patients suffering from paralysis agitans, a slowness of movement in the change of direction of the axes of the eyes and a widening of the palpebral fissure, giving a staring expression to the eyes and fixity to the gaze.

Orzechowski [21] also described a condition that he termed "opsoclonie" as a continual state of agitation in which the eyes pass through rapid and unequal movements, usually in the horizontal plane. These movements may occasionally be interrupted by crises of upward gaze of short duration. The movements are more apt to appear at the inception of movement, resulting in a change of position of the eyeball either voluntary or reflex. These movements are most intense at the beginning of the movement and tend to disappear when the direction of gaze becomes fixed. This condition is associated in his cases with other signs of involvement of the nervous system, such as polyclonies, myokymias

and tremors of the fingers, chin, lips, eyelids and forehead. These movements are increased by attention and emotion and are accompanied by some signs of cerebellar involvement. He attributed the opsoclonia to disturbances of the basal ganglions and likened them to phenomena that arise in the course of Parkinson's disease. Occasionally movements similar to those seen in the face, lips, chin, tongue and other portions of the somatic musculature may be seen in the eyes.

In all probability the basal ganglions have a definite relationship to ocular movements. Alajouanine [22] reported the case of a patient showing the syndrome of Parinaud, a loss of vertical movement in which there were other evidences of diffuse extrapyramidal hypertonia affecting the eyes and associated with certain pseudobulbar difficulties. He stated that no actual paralytic disturbance was present, and he attributed the pathologic symptoms present to an interference with tone, a true hypertonia of the ocular muscles, the entire syndrome apparently being due to encephalitis.

In the present state of our knowledge of extrapyramidal physiology, it is impossible to discuss these hyperkinetic phenomena categorically, and impracticable to attempt any kind of anatomic or physiologic localization. The diseases of known extrapyramidal origin, particularly those attributed to striatal pathologic changes, are characterized by deviations from the normal in the realm of oculomotor control in regard both to tonal changes and to the production of abnormal involuntary movements. In certain instances, motility disorders may be seen in the eyes that are in agreement with the particular type of motor disorganization that is present in the rest of the body musculature, and it can at least be presumed that the disturbances seen in the muscles of the eyes and in other muscles depend on analogous pathologic changes.

Oculogyric Crises.—These oculomotor disturbances have been found chiefly since 1923 in the large number of persons afflicted with epidemic encephalitis. These patients are characterized by deviations of gaze that are usually upward and to the left, although they may take place to the right or downward. The pathologic substratum for the production of these forced conjugate deviations is still a matter of much debate, certain authors believing that they are due to cortical pathologic processes and still others that they are psychogenic in origin.

Wimmer,[23] in describing these abnormal movements, compared them with the tonic movements that are seen in epileptic convulsions; he used

22. Alajouanine, T. H.; Delafontaine, P., and Lacan, J.: Fixité du regard par hypertonie, prédominant dans le sens vertical, avec conservation des mouvements automatic, reflexes, aspect spécial de syndrome de Parinaud par hypertonie associée à un syndrome extrapyramidal avec troubles pseudobulbaire, Rev. neurol. **2**:410, 1926.

23. Wimmer, A.: Tonic Eye Fits, Acta psychiat. et neurol. **1**:173, 1926.

the term extrapyramidal epilepsy or brain stem cramp. He quoted C. and O. Vogt as predicating an extrapyramidal or striatal center for the conjugated movement. He expressed the belief that the association of other extrapyramidal manifestations in the postencephalitic syndrome may support the view that these tonic eye fits are associated in their causation with other manifestations of extrapyramidal disease. According to Bing and Schwartz,[24] these manifestations are neostriatal in origin and represent a release phenomenon, an uncontrolled activity of the subcortical centers resulting from a removal of the inhibition which is exercised by the newer portion of the basal ganglions over the older representatives. Wimmer [25] also spoke of the possibility of a cerebellar origin for these movements, a lesion in the cerebellum providing abnormal stimuli that act on the striatum and produce these abnormal movements.

Kennedy,[25] in describing the ocular disturbances in encephalitis, attributed the ocular spasms, the clonic contractions of the eyelids and the oculogyric crises to a pouring down of impulses from the basal ganglions activating the oculomotor and associated centers and producing oculomotor disturbances similar to the other abnormal involuntary movements and the hypertonia so characteristically found in postencephalitic disease and attributed to the uncontrolled activity of the striatum.

Pardee [26] reviewed these paroxysmal oculogyric crises and considered them to be due to disturbances in the striatal mechanism, producing their results through the posterior longitudinal fasciculus and the anterior corpora quadrigemina.

Tinel and Baruk [27] described these crises and drew resemblances between them, migraine and hysteria. He stated that he believed they are due to mesencephalic angiospasms.

In regard to the etiology of these oculogyric crises, the essential causative factors have been postulated to exist in other regions also, and this will be more fully dealt with in the consideration of the ocular dysfunctions that occur as a result of cortical disease.

THE CONTROL OF OCULAR MOVEMENT BY THE CEREBRAL CORTEX

Before undertaking a detailed examination of the actual control of ocular movement by the cortex, it might be advantageous to survey

24. Bing, R., and Schwartz, L.: Movements of the Eyes in Parkinsonism, Encéphale **20**:150, 1925.

25. Kennedy, F.: Ocular Disturbances in Epidemic Encephalitis, Arch. Ophth. **1**:346 (March) 1929.

26. Pardee, I. H.: Paroxysmal Oculogyric Crises in Parkinsonian Encephalitis, Tr. Am. Neurol. A., 1927, pp. 359-367.

27. Tinel, J., and Baruk, H.: Crises hypertoniques oculigyres d'origine encéphalitique, Encéphale **21**:778, 1926.

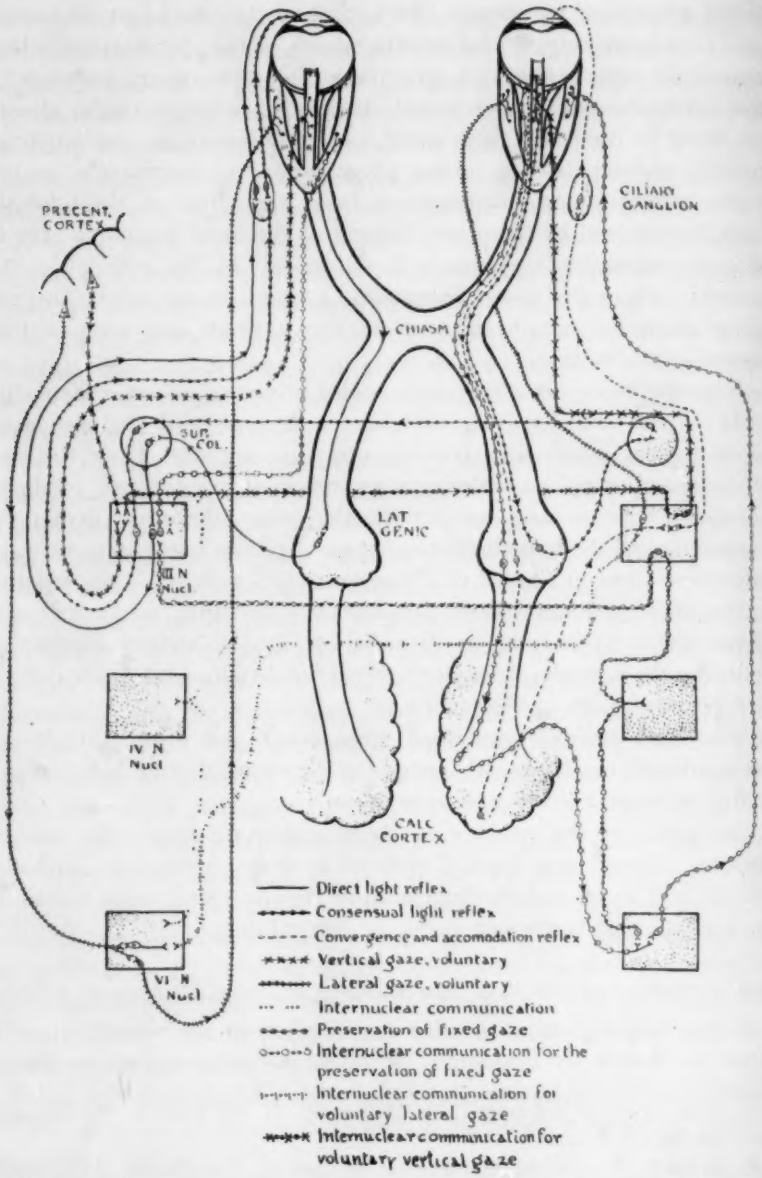

Fig. 8.—Diagram illustrating various pathways in the pupillary and oculomotor mechanism.

briefly the general evolution of vision and of eye movements that has taken place in the phyletic scale. In considering the lower forms of the mammalian order, it is easily recognized that the great majority of them possess eyes that are situated on the lateral aspect of the head separated by the projecting head and snout. In many of these animals the eyes are so placed that there is no overlapping of the visual field, therefore no binocular vision and therefore no need for the conjugation of the movements of the eyes. The movement of the head and neck and sometimes of the entire body is substituted for the movement of the eyes. In the primates and man, the forward position and great mobility of the eyes in conjugated movement enables the individual to perceive the entire range of vision in front of him without moving the head, the macula being brought to bear directly on the object by the movement of the eyes alone.

According to Collins,[28] binocular vision is seen particularly in those forms that have a carnivorous dietary. In these animals, including not only the mammals but also the carnivorous fish, amphibia and birds, the eyes, instead of being situated on the lateral surface, have moved forward in varying degree to a situation more on the front of the head in order to provide for an overlapping of the visual fields. It may be presumed that this characteristic in these carnivorous animals is in the interest of facilitating and rendering more efficient the pursuit of their food supply, which is animate and possesses movement, thus enabling it to escape from its pursuers.

In the study of the human embryo we find that ontogeny repeats phylogeny not only in the development of the visual apparatus, but also in many other parts of the nervous system. In the human embryo, the optic vesicles arise as evaginations from the neural tube in the vicinity of the differentiation of the telencephalon and the diencephalon. This evagination from the neural tube forms the basis for the optic apparatus. Originally this evagination takes place from the lateral surface of the embryo, the axis of the two optic vesicles being at right angles to the long axis of the neural tube. If this condition persisted, the eyes would be situated on the lateral surface of the head. In the course of embryologic development, however, the axis of the optic stalk and vesicle gradually undergoes a shifting forward, so that in the later embryonic stages the axis of the optic vesicles and stalk lies almost parallel to, but slightly divergent from, the axis of the neural tube itself.

Wood Jones [29] expressed the belief that this transition of the eyes in the course of phylogenetic development from the lateral aspect to the front of the head is brought about by the recession of the snout trans-

28. Collins, E. T.: Arboreal Life and the Evolution of the Human Eye, Philadelphia, Lea & Febiger, 1922.

29. Jones, F. Wood: Arboreal Man, London, E. Arnold, 1916.

forming a long face with the eyes at the side into a flat face with the eyes situated anteriorly. The recession of the snout apparently follows the reduction of the necessity for the sense of smell and for the use of the jaws and teeth in attack and defense. This diminution in the importance of the snout and jaws takes place pari passu with the development of the fore limbs, the freeing of the fore limbs from the necessities of locomotion and the substitution of the hands for the purpose of exploration of the environment and for the pursuit and capture of food. This transition is particularly favored by the assumption of arboreal life in which existence in three planes of space is substituted for that carried on in two planes of space, as is the case with animals that live to a large extent on the surface of the earth. The development of conjugate movement of the eyeballs and of the consensual light reflex takes place simultaneously with the migration forward of the eyes and the acquisition of overlapping fields of vision that provides for binocular vision.

The acquisition of binocular vision is accompanied by an increasing proportion of nondecussating fibers in the optic tract. In rodents, which have no conjugate movement and no consensual light reflex, only a few of the optic fibers decussate. This is true also of the ungulates. In the carnivores, a certain number of the fibers from each retina pass to the homolateral cortex; observations following experimental investigation of vision in cats shows that they apparently suffer from something that is similar to a homonymous hemianopia, and in these animals a considerable number of nondecussating fibers equally distributed throughout the optic tract may be found.

The evolution of binocular vision results in a segregation of the fibers that pass to one side of the brain or to the other according to the part of the retina from which they take their origin. The fibers from the temporal portion of the retina of one eye and the nasal portion of the retina of the other eye pass to the corresponding side of the brain and vice versa, so that each side of the brain, instead of receiving impressions from the entire field of vision of the contralateral eye, is stimulated by rays of light that are reflected from objects situated in the contralateral half of the horizon.

True central vision appears only in the primates, although areas of more highly developed retinal sensitiveness are apparently present in some of the lower forms. Also in the primates, for the first time, is found an actual power of convergence and accommodation. In the acute central vision that is present in primates, particularly in man, there is apparently a bilateral representation of each macula in the occipital cortex. This is brought about either through the fact that fibers from each macula proceed to the occipital lobe of both hemispheres, thus providing a crossed and an uncrossed connection between this portion of the retina and the occipital lobe, or, as is maintained by Cajal and Kolliker,

this bilateral representation is produced by a bifurcation of the optic fibers in the optic chiasm. The present preference would seem to favor the bilateral representation of the macula through crossed and uncrossed systems of fibers, rather than through an actual bifurcation of the fibers in the chiasm. Whatever may be the cause, the result is the sparing of central vision in lesions that may be so placed as to produce a bilateral hemianopia. This arrangement enables the brain to obtain views of any object fixed on, from two different angles, this double impression providing for the visual perceptions of size, shape, spatial relationships, stereoscopic effect and the sense of distance.

The relationship of the palpebral fissure to the area of the cornea has also undergone a progressive increase in the phyletic scale, and man presents the maximum in this relationship. In all probability, this greater proportion between the width of the palpebral fissure and the size of the cornea is to provide for an increased range of movement and of vision.

Careful observation of the gradual acquisition of coordinated movement, not only in the conjugated movements of the eyes but also in association with movements of the eyelids, were made by Preyer.[30] According to his investigation, the movements of the eyeballs may or may not be coordinated at birth. By certain observers, the coordinated movements that do occur are considered to be only those of chance coincidence in the ordinary movements of the eyes in all directions.

Collins stated that the vertical movements of the eyeballs together are present shortly after birth, which he adduces as evidence that the deviations of the vertical gaze are of older phylogenetic history than are the lateral deflections, and that they represent the more or less instinctive responses of the eyes to habitual or usual sources of danger in man's previous ancestors. He bases this hypothesis chiefly on the arboreal habitat, and predicates the cause of these movements on the probability or frequency of attacks on primitive man or the primates from sources which were more usually situated above or below the individual than to either side.

According to Preyer's experiences in the new-born, the eyes usually move independently of each other. He has observed that the head and eyes frequently move in opposite directions. This is a movement that is almost impossible or at least difficult to produce in the voluntary control of the head and eyes in the adult human being. He has also noted unconjugated movements of the eyeballs in infants while they are asleep.

Schoeler [31] stated that no perfectly correct fixation takes place at any time until after the tenth day, and that up to the fifty-fifth day or

30. Pryer, W.: The Senses and the Will, New York, D. Appleton & Company, 1893.

31. Schoeler, quoted by Preyer (footnote 30).

so irregular, uncoordinated and unconjugated independent movements of the eyeballs are seen. The movements of the eyelid may also be alternate up to and some time after the eleventh day, the elevation of the eyelids not being synchronized with the eyeballs and often in an opposite direction. This coordination is not perfected until at least the second month of life. The association of the elevation of the forehead with the eyeballs when looking upward does not definitely take place until at least the ninety-eighth day of existence, and this association is apparently a learned one. The reflex closure of the eyelids to light apparently is innate and takes place at birth, whereas defense movements do not take place until the seventh or eighth week.

Conscious vision has played the decisive rôle in the regulation of the eye movement. The stimulus to conjugated movement of the eyes does not appear until the function of vision begins to be active. The actual conjugation of the eyes together is apparently a learned reaction, and the development of homonymous areas of the retina takes place only after conscious vision and conjugate movement have developed to a considerable extent. Fixation of gaze is not present in infants, but even as early as the first day the eyes may be seen to be turned toward a light. This has not been interpreted as a true turning of gaze, but rather as a reflex response to the stimulation of the light. Some conscious direction of gaze seems to make itself apparent at about the fourteenth day of life, but the definite following of objects with the beginning appearance of intelligence and appreciation in the movements of the eyes does not take place until toward the end of the first month, and even then the object must pass slowly through the field of vision in order to be followed by the eyes. This does not necessarily predicate that the volitional connections are already established at this early date, for there are centers subcortical in position that seem to exert considerable influence over the fixation of gaze independent of volitional activity. The actual transition from reflex turning to conscious fixation of gaze is not definitely established. It does not seem to take place before the twelfth week, and it is not before the hundredth day that quick lateral movement begins to appear. The function of accommodation does not appear much before the ninth week, and the appreciation of size, form and distance apparently is delayed considerably, at least beyond the eighteenth week, as is evidenced by the faulty reaching of infants at objects that are situated at a considerable distance from them. Accurate conjugation of lateral movements of the eye apparently does not become well established before the end of the first six months.

In sleep, many symmetrical and unconjugated movements of the eyeballs may take place. The eyes may present a tendency toward convergence or divergence. Unassociated movements in which one eye

moves in one direction and the other eye moves in the other direction are common in cases of suspended consciousness, in profound drunkenness, and under certain circumstances similar movements are seen in persons who are not exercising the function of vision for the purpose of fixing the visual axes on any given object.

The actual cortical control of the ocular movements has been definitely determined to reside principally in the caudal extremity of the second and third frontal convolutions, where they are contiguous to the precentral convolution. In this vicinity there is apparently situated a net of points stimulation of which produces eye movements in one or another direction, and it is from this area that the chief volitional control over the ocular movements takes its origin. On account of the fact that even with severe cortical lesions, oculomotor control, already paralyzed, is often slowly regained, it is probable that there is some degree of bilateral innervation of the gaze movement by each hemisphere. The deviations that take place as the result of cortical stimulation are always in the production of conjugated movements of the eyeballs in the direction of gaze. This means that in the cortex are contained patterns by which the oculomotor nuclei and segments of nuclei controlling various bilateral combinations of the eye muscles may be simultaneously activated in producing conjugated gaze deviation in one direction or another. These gaze movements are concerned in upward, downward and lateral gaze and convergence.

In 1863, Prevost [32] recognized the paralysis of lateral gaze that results from cerebral accidents, and he described the condition as characterized by a loss of movement away from the side affected, as a general rule unaccompanied by double vision.

The oculogyric movements are usually associated with movements of the head, and the centers for oculogyric and cephalogyric movement are in close contiguity. The association of oculogyric and cephalogyric movements is close. The parallelism of the optical axes is maintained in no matter what position the head may be placed. The extent of movement of the head in connection with the deviation of gaze is commensurate with the necessity for the gaze deviation. In the usual movement of the eyeballs, it is common to find the lateral ocular movements taking place without any movement of the head unless the object that attracts attention or must be seen is placed so far to the side that the head must be turned for it to be clearly observed. In the vertical movements of the eyeballs it is much more usual to find them associated with upward and downward movements of the head. This is particularly true in the oblique movements which when carried out by the eyeballs alone result in rather disagreeable sensations, so that when an object

32. Prevost, quoted by Wilbrand and Saenger (footnote 1).

obliquely above or below is to be viewed directly, the movements of the eyeballs are largely substituted for by movements of the head in the oblique upward or downward direction.

On stimulation of the caudal portion of the second and third frontal convolution, movements of the eyeballs laterally have been obtained with complete regularity by many investigators, including Monakow, Mott, Sherrington and Schuster and others.[33] Upward, downward and convergence movements of the eyes, either isolated or associated with head and eyelid movement, have also been seen. Stimulation of the calcarine area has produced lateral and convergence movement.

Russell,[34] working in conjunction with and under the direction of Sherrington, obtained interesting results from stimulation of the second and third frontal convolution under various conditions. It was found that stimulation of this region almost universally produced lateral deviation of the eyes. It occurred to these investigators that if the muscles for lateral gaze were cut it might be possible to produce other deviation of gaze by stimulation of this center. Accordingly, a large series of experiments was performed in which in succession various combinations of muscles were rendered inoperative by section; on stimulation of the cortex it was then possible to produce conjugated movement in every direction when the proper muscle was sectioned in an appropriate animal. These investigators therefore concluded that the second and third frontal convolutions were responsible for the cortical control of conjugated movement of the eyes in every direction, but that the lateral movements were so much more usual and so much more commonly exercised that this dominance overshadowed the control of the other conjugated movements of the eyeballs. By means of repeated experiments, they were able to determine subsidiary areas within this general region which seemed to provide definite control over the conjugated gaze movement in all directions.

Bramwell,[35] in reviewing the general situation concerning the upward movement of the eyes, emphasized the fact that movements and not muscles are represented in the cortex. He also pointed out that in the convulsive seizures the eye movement manifestations are those always of lateral movement, that they are associated with cephalogyric movements also, and that vertical movements are seen only exceptionally in mild convulsive petit mal episodes.

The volitional control of the ocular movement is dependent, as are all other movements on the motor incentives and motor purposes that

33. Monakow, C. V.: Die Lokalization im Grosshirn, Munich, J. F. Bergmann, 1914.

34. Russell, J. S. R.: An Experimental Investigation of Eye Movements, J. Physiol. **17**:1, 1894-1895.

35. Bramwell, E.: The Upward Movement of the Eyes, Brain **51**:1, 1928.

are transmitted to the intermediate precentral region at the caudal extremity of the second and third frontal convolutions from all other areas of the cortex. The impulse for movement arises as the direct result either of incoming impressions or of impressions that have been received in the past, stored up in the mnemonic areas of the cortex, and, being again brought up into consciousness, produce volitional movement. This impulse association takes place within the intermediate precentral area and is transmitted by short association fibers directly to the giant pyramidal cells of the precentral convolution which are connected with the various oculomotor nuclei or centers. These cells of Betz give rise to axons that pass from the cortex into the corona rădiata of the white matter and enter the genu of the internal capsule. Reaching this point, these fibers turn downward and pass through the internal capsule together with the other corticobulbar axons which together form the aberrant pyramidal tract. Separate contingents from the tract are given off at different levels within the neuraxis. The subthalamic contingent which is concerned principally with vertical movement, and therefore associated with the movements of the upper eyelid and the frontalis muscle, leave the aberrant pyramidal system in the subthalamus, pass directly backward in its substance, perhaps in the fields of Forel, and reach the superior colliculi. In these structures the fibers are redistributed either by means of a center for vertical gaze in or near the superior colliculus or pass through it to the nuclei that govern the muscles associated together in the conjugate movement of vertical gaze.

The peduncular contingent of the pyramidal tract that is destined for the supply of lateral movement leaves the corticonuclear tract as two bundles of fibers, the pes lemniscus superficialis and the pes lemniscus profundus in the midbrain and upper pontile region, respectively, descend for a short distance in the mesial fillet and then proceed dorsally in the tegmentum to the center for lateral movement, which is situated in or close to the nucleus of the abducens nerve.

The course of the fibers concerned with convergence is not definitely known. Convergence movements have been produced by stimulation of the center in the frontal lobe. They have also been obtained by stimulation of the occipital cortex on the lateral convexity of the occipital pole. As convergence is closely associated with fixation of gaze, it is possible that the occipital as well as the frontal cortex is able to control, initiate or maintain this type of gaze deviation.

PRESERVATION OF FIXED GAZE

Control by an occipital center over the maintenance of fixed gaze is a phenomenon for which a definite reason can be seen and for which there is considerable clinical evidence. It has been repeatedly shown that patients suffering from paralyses of lateral gaze are capable of a

greater or less degree of movement of the eyeballs in the voluntary paretic zone when the movements are not the result of volitional effort but are carried out for the purpose of maintaining fixation of gaze. The advantage of a mechanism by which the visual cortex is enabled to maintain directly the two maculae fixed on a moving object needs no particular argument. This function appears to be supervised not only by the frontal, but also by the occipital lobe, and different pathways are utilized in transmitting this control. Movements of the eyeballs in the zone paralyzed for volitional conjugate deviation of gaze can be demonstrated by requiring the patient to fix on an object as seen in the nonparalyzed zone and then to follow the object into the field of activity of the paretic muscles. Another method by which the preservation of fixed gaze in the presence of paralyzed voluntary conjugate deviation can be demonstrated has been pointed out by many observers. Some years ago I [36] observed a patient who presented complete paralysis of gaze toward one side, but when the head was passively rotated by the examiner after the gaze was fixed on an object, the eyes deviated into the zone of paralyzed voluntary movement, thus showing that the pathways for volitional and fixed gaze control are separate and that fixation of gaze does not use the same machinery as does the volitional direction of gaze. The connection for this occipital oculogyric control must of necessity be provided by a corticofugal group of fibers leaving the occipital oculogyric center, passing through the corona radiata over the occipital radiation into the white matter of the hemisphere and then to the oculogyric centers by way of the posterior longitudinal fasciculus of the tectobulbar tract. The control of the ocular movements exercised by the parietal and temporal lobes is not nearly so well known. Mention of these movements as produced by stimulation or by lesions situated in these localities is scanty, and the physiologic character of the connection is not well established. If the connections are valid, they are probably for the purpose of providing associations between the somesthetic or auditory impressions and movements of the eyeballs.

UNUSUAL VOLITIONAL CONTROL OF THE MOVEMENTS OF THE EYES

Many years ago, Hering established the law known by his name, namely, that cortical innervation of the ocular muscles is never unilateral but that proportionate innervation of both eyes takes place in all movements initiated by the cortex.

Wilbrand and Saenger, although they stated that unilateral movements of the eyes are impossible as the result of voluntary innervation,

36. Riley, H. A.: Report of a Case of Conjugate Deviation of the Eyes with Astereognosis, Arch. Ophth. **48**:4, 1919.

still described movements of divergence and of unilateral raising and lowering of the eyeball and also of an abduction of one eyeball alone. They stated, however, that most of these unilateral movements are dependent on a weakness in conjugated movement and that some sort of disturbance may be discovered in one or another of the oculogyric muscles.

In discussing unusual voluntary control over the oculomotor mechanism, Bertein [37] described a voluntary nystagmus and a voluntary dissociation of ocular movement. In this case the patient was found to be capable of producing rapid voluntary pendular movements of the eyes in a conjugated fashion to one side or the other. The movement was not phasic, as in the case of vestibular nystagmus, was of equal length in its two components and could be maintained for only a limited length of time. The patient could also produce a unilateral internal movement of one or the other eye on voluntary effort.

Spiller [38] reported the case of a patient who was capable of unilateral (unassociated) innervation of the ocular muscles. In this patient the conjugate movements were normal, except for convergence; in addition to this, he could fix one eye on the examiner and deviate the other eye outward into the external canthus. The movement could be produced by either one or the other eye, and the deviated eye could be brought back to the point of fixation slowly or rapidly. The vision of this patient was normal in every respect, except that he possessed monocular vision. He passed through an attack of epidemic encephalitis seven years previously, the persistent manifestations of which were rolling or twisting movements of the tongue, a tremor of the lips, chin and right hand, together with a tendency to fall asleep in the day time. Spiller called attention to other apparent contradictions of Hering's law, and stated that as unilateral movement of the eyeball is possible in some of the lower animals this type of cortical control must at least be potential, and that occasionally a reversion to a more primitive type of movement is possible in the human being.

Ayer [39] reported the cases of two brothers, each possessing normal vision, in whom there was discoverable a monocular voluntary control in adduction. One eye being maintained on the point of fixation, the other one could be moved slowly inward and returned to the conjugate position. He was not certain whether or not this was a true monocular voluntary control due to separate ocular muscle cortical representation.

37. Bertein, M.: Nystagmus volontaire et dissociation des movements oculaires, Lyon méd. **143**:31, 1929.

38. Spiller, W. G.: Unilateral (Unassociated) Innervation of the Ocular Muscles, Tr. Am. Neurol. A., 1927, pp. 368-385.

39. Ayer, J. B.: Voluntary Dissociated Movements of the Eyes, J. Nerv. & Ment. Dis. **69**:69 (Jan.) 1929.

DISTURBANCES IN CORTICAL CONTROL OF THE OCULAR MOVEMENTS

Lesions producing disturbances in the ocular movements by interfering with the cortical centers or the connections between these centers and the brain stem nuclei always affect gaze movements and are not concerned with single eye muscles. Here attention may again be drawn to the criteria in connection with the discussion of the nuclear palsies and their differentiation from cortical lesions. These gaze or conjugate deviation lesions are of a purer character the further away the lesion is from the brain stem and the nearer it is to the cortex. With lesions in these pathways, associated nuclear disturbances are sometimes found which obscure the pure deviation paralyses or hyperkinesias. The individual muscles are still capable of movement in the other directions of gaze and in other isolated movements, and they are also still subject to the reflex control exerted principally by the vestibular and the trigeminal connection; under certain circumstances they are capable of the paradoxic movement that may occasionally be seen with the deviation of gaze concerned in the preservation of fixed gaze.

Lesions situated principally in the cortex and more rarely along the course of the aberrant pyramidal bundles may be either of an irritative or of a paralytic character. In the irritative type spasmodic deviations of gaze are found that are exemplified particularly by those associated with convulsive seizures. Similar less extensive but more constant gaze spasms may be caused by foci of irritation as the result of inflammation, infection, neoplastic pressure, meningeal thickening as the result of trauma or by some exogenous or endogenous toxemia. When the process is progressive, this irritative stage is usually followed by a paralysis in which deviation of gaze in one or another direction becomes impossible. Lesions affecting the oculogyric pathways in the peduncular region are capable of producing interference with lateral gaze, either singly or bilaterally, according to the position of the lesion. They are often associated with weakness of cephalogyric control and result in a deviation of the head in the same direction as the deviation of gaze. These paralyses may also be associated with other motor or sensory manifestations, and the exact level diagnosed as the site of the lesion will depend on the correct appreciation and determination of the associated evidence of neuraxial involvement. In the early stages of pontile lesions, the irritative phase may first be seen deviating the gaze toward the affected side only to be replaced later by the paralytic gaze disturbance in which the eyes are drawn away from the side of the lesion. The reversal of the relationship between the side of the lesion and the direction of the gaze in pontile lesions is, of course, due to the decussation of these pathways shortly before their destination is reached.

The oculogyric crises so common in the parkinsonian form of epidemic encephalitis have been exhaustively investigated by Jelliffe.[40] They are frequently associated with states of fatigue, psychomotor activity, compulsions, phobias and other evidences of cortical or subcortical stimulation, such as fear of going blind, or of visual hallucinations either simple, such as seeing red, or of a more complicated character. These evidences of psychic activity are so constant that the possibility or probability of the impulses concerned in these crises originating in the cortex or near to the cortex must be considered. As pointed out by Wimmer,[23] Barkas [41] and Bailliart and Blum,[42] the association of fatigue, psychic strain and loss of voluntary power together with other mental manifestations must make such a consideration necessary. The psychic content of these oculogyric crises is described in detail by Stern,[43] who emphasized the fact that no actual ocular paralysis need be present, that usually a weakness of will and of the mental processes is present, that associated anxious compulsions appear, and that consciousness may be more or less involved. He concluded that the crises result from the reactivation of affective states incorporated into the soul life and evoked by trauma or infection as symbolic representations of cortical or subcortical activity.

McCowan and Cook [44] reviewed the situation concerned in these oculogyric crises, and stressed the associated psychic phenomena of compulsive thought, feeling of dying, of going blind, that the eyes will never come down, screaming, crying, sighing, etc. They expressed the belief that the foci productive of these oculogyric spasms are not situated in the lower centers but in the associational pathways of the cerebral hemispheres. They pointed out the evident psychic components, the tendency of the crises to occur at certain times of the day or on certain days, and that they are subject to psychic stimuli not only in their production but also in their suppression.

40. Jelliffe, S. E.: Oculogyric Crises as Compulsive Phenomena in Postencephalitis: Their Occurrence, Phenomenology and Meaning, J. Nerv. & Ment. Dis. **69**:59, Jan.; 165, Feb.; 278, March; 415, April; 531, May; 666, June, 1929; Psychologic Components in Postencephalitic Oculogyric Crisis, Arch. Neurol. & Psychiat. **21**:491 (March) 1929.

41. Barkas, M. R.: Tonic Spasm of the Eyes in Conjugate Deviation, Lancet **2**:330, 1926.

42. Bailliart, P., and Blum, J.: Crises oculaires toniques latérales et paralysie de la convergence d'origine encéphalitique, Bull. Soc. d'opht. de Paris, 1927, pp. 66-70.

43. Stern, F.: Ueber psychische Zwangsvorgänge und ihre Entstehung bei encephalitischen Blickkrämpfen mit Bemerkungen über die Genese der encephalitischen Blickkrämpfe, Arch. f. Psychiat. **81**:522, 1927.

44. McCowan and Cook: Brain **51**:285, 1928.

Critchley[45] drew attention to the frequent coexistence of the crises with associated spasmodic movements of the brachial, facial and respiratory musculature. He drew attention to the fact that the crises are followed by sleep, and therefore he places them in the same category as that occupied by the narcolepsies.

Delbeke and van Bogaert [46] considered this condition to be due to a state of extended inhibition, a mixed condition similar to that of cataleptic sleep and the dreamy states of uncinate convulsions. They emphasized the fact that the crises may be only a part of a complex psychic state with ocular disturbances as the only evident external manifestation. They pointed out the frequent association of anxiety, obsessions, repressions, hallucinations and psychic inhibition of variable intensity.

With the views of the origin of these crises so markedly at variance, it would appear at least wise to forego any exact localization at the present time and conclude that they present constituents that bear the characteristics of both cortical and subcortical (striatal) origin. Marinesco and Radovici [47] look on these disturbances as hysterical, but this view would seem to deserve little consideration on account of the wealth of organic evidence which these patients almost invariably disclose on careful neurologic examination.

FUNCTIONAL DISTURBANCES OF OCULOMOTOR CONTROL

Wilbrand and Saenger reported a group of oculomotor phenomena that they attribute to hysteria, including movements that are distinctly nuclear. From our knowledge of the production of hysterical phenomena it would seem wiser to restrict hysterical manifestations to those that can be produced by the higher centers alone, including perhaps in these, the basal ganglions.

The manifestations of hysteria are principally restricted to unilateral or bilateral ptosis, to cramps and deviations of conjugate gaze, paralysis of any form of conjugate gaze or to a complete immobility of both eyes. The determination of a functional oculomotor disturbance must depend on the identification of a sufficient psychic mechanism and on the recognition of other evidences of hysterical involvement.

45. Critchley, A. M.: Ocular Manifestations Following Encephalitis Lethargica, Bristol Med.-Chir. J. **45**:113, 1928.

46. Delbeke, R., and van Bogaert, L.: Le probleme générale des crises oculogyres au cours de l'encéphalite épidémique chronique, Encéphale **23**:854, 1928.

47. Marinesco and Radovici, quoted by Wimmer (footnote 23).

Ophthalmologic Review

THE DIFFERENTIATION OF THE OCULAR MANIFESTATIONS OF HYSTERIA AND OF OCULAR MALINGERING*

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The differential diagnosis of these two allied conditions, the ocular manifestations of hysteria and of ocular malingering, cannot be covered in a comparative review by a mere cataloging of facts, of more or less authoritative statements and of clinical observations, not to mention various theories and deductions. Even a bibliography must be given with an apology; it cannot be outlined with meticulous detail. The literature is voluminous, and the ramifications of allied interests are extensive. Also, this review is not a dissertation on the ocular manifestations of hysteria, save for its application to the second condition. De Schweinitz has long since covered hysteria in a complete manner, but little else can be said on the subject.

An attempt will be made first to differentiate one from the other by an understanding of each, and then to compare the conditions as two definite pathologic entities. The various tests for the detection of simulation will not be considered. They would be irrelevant here. A word of warning must also be expressed as to the value of these tests. They too often have the element of pain, of surprise and of "trapping" the patient. In many instances, the person with true hysteria will respond to the tests in the same manner as the simulator. There is no doubt that they have a definite place in semeiology and in diagnosis, but as great an importance lies in the negative characteristics of each condition. Todd and Charcot first pointed this out by their invention of certain Greek words to describe some of these, i. e., helicopode versus helcopode gaits, etc.

The history of hysteria dates from the earliest years of medicine; its name demonstrates this conclusively. Plato (427-347 B. C.), as quoted by Stewart, even gave his idea of its etiology. "The matrix is an animal that longs to generate children, . . . etc." As customs, habits and standards of living changed, so also did its symptomatology, and with these the attitude of the layman and of physicians underwent complete reversals. It is a far cry, however, from the ὑστέρα of early Greece to the present day understanding of this interesting condition.

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Malingering, or simulation, has been practiced as long if not longer. A quotation from the *Tatler* (1709) is rather suggestive of the age of this almost ubiquitous trait. Both conditions are as interesting as is any clinical entity in medicine.

HYSTERIA

Charcot and Janet, in the beginning, then later, White and Prince, Starr and Jelliffe, and more recently Jung, Stekel and Adler, with Freud, have carried hysteria from the early realm of descriptive psychiatry into the present day field of interpretative psychiatry, hence with an entirely different attitude toward this psychosis. This is so in spite of, and regardless of, the various individualistic theories as to the content of the psychosis these various men and schools may have. (To mention all neurologic as well as the various mechanism theories is unnecessary as well as impossible.) It is sufficient at this time to adhere to one outstanding school and thus build up an adequate working fabric.

The hysterical make-up, from the standpoint of personality defects, is manifested by a poor synthesis of the personality, which makes it possible for certain groups of ideas to drop from an effective association (hence dissociation) with the main portions of the personality. They occupy a region first called and explained by Janet as the subconscious, and there existing more or less independently produce their effects irrespective of attempted corrections from the rest of the personality.

Janet, as outlined by Hart, based this dissociation of the personality on a doctrine of psychologic tension—a property he described as analogous in its reactions to atmospheric pressure—the variations being produced by constitutional or emotional causes. When it is high, mental processes are integrated with comparative ease, and efficient adaptation to reality is attained. When it is low, the required integration is impossible, and dissociation of some kind occurs. Wholey has illustrated this, which can occur, in another manner. When situations of extreme difficulty arise there are but two alternatives—fight or flight. If, however, the obstacle cannot be met by a person—say one hysterically constituted—a compromise situation will develop. He can take on some bodily incapacity as a legitimate excuse for abandoning the struggle, thus squaring his own conscience (?) and providing against the censure of public opinion. Such a reaction represents a partial break in mental control, in other words, a shutting off of recognition by the psyche of some organic function (as vision) and absolving the person from further struggle. In another case in which consciousness as a whole is affected, there is a greater cleavage of psychic function as memory, volition, etc. It is an almost instinctive reaction on the part of a faultily

equipped person. His hysterical conduct may be infantile and ineffectual, but it represents that particular person's most efficient way of meeting a situation. These manifestations are always symbolic, harking back to the specific psychic conflict.

It is true that Janet's theory is rather incomplete for the psychologist, but at that time it was the first basis for much later development. As Hart said, while the study of dissociation takes one toward the understanding of a patient, it leaves one with the problem of explaining this dissociation. Again to return to Freud, for regardless of the point of view a person may have as to the validity of some of his doctrines, recent advances are unquestionably due to him. With him, psychoneuroses develop as a result of a conflict between forces emanating from sex instincts and personality forces, the libido and the ego. As he stated, "the failure of adjustment arises when the ego loses its capacity to deal in some way with the libido." Further, his conception of the unconscious, the preconscious and the conscious is a further integral part in the theories on which the psychoneurosis of hysteria is based. Relative to dissociation, Freud stated that (according to Starr) these ideas are out of harmony with the rest of the personality, that they represented ideas which were in conflict with the ideas forming the consciousness of the person, therefore repressed, and with conversion as a result. Repression then, is with Freud the fundamental factor or the basis of hysterical manifestations, an active and not a passive mental factor which tends to put out of the conscious mind these certain inacceptable groups of ideas.

The psychosexual side of the problem received a most intensive investigation at the hands of the Zurich psychiatrist, E. Bleuler, shortly after Freud promulgated his tenets on the origin and development of this psychosis. Bleuler said he openly faced the sexual theories incredulous and bewildered, but since, by his observations, he has substantiated them in their whole scope. Occasionally this factor is to the more casual observer not outstanding as in the case quoted by Weinberg (see Smeltz) of the college student who developed a paralysis of his left arm while visualizing himself captain of a great football team and at the bottom of a scrimmage heap. He did not like his school work, and herein lies the application of the conversion theory. Recovery occurred in fifteen minutes. Wholey's cases, on the other hand, show readily the infantile sexuality in hysteria symptomatology.

In 1910, while lecturing at Clark University, Freud made two statements applicable herein relative to the psychoanalysis of hysteria and the other neuroses. While these statements are taken from their proper setting in this lecture, they serve most admirably here, for to be able to discriminate between the ocular manifestations of hysteria and

the manifestations of ocular malingering demands a fair understanding of each. "Our hysterical patients suffer from reminiscences, their symptoms are the remnants and the memory symbols of certain (traumatic) experiences." A flight from unsatisfying or painful reality into what one calls disease, on account of its biologically injurious nature, but which is never without an individual gain in pleasure for the patient, takes place over the path of regression. In the second instance, Freud stated, "we come to the conclusion, from working with hysterical patients and with other neurotics, that they have not fully succeeded in repressing the idea to which the incompatable wish is attached." (The wish, the traumatic and distressing experience, the erotic impulse components, the reminiscences, are all in fact repressions, and hence one in effect.) They have driven it out of consciousness and out of memory, and apparently saved themselves a great amount of psychic pain, but in the unconscious the suppressed wish still exists, only waiting for its chance to become active, and finally succeeds in sending into the consciousness, instead of the repressed idea, a disguised and unrecognizable surrogate-creation (Ersatzbildung) to which the same painful sensations associate themselves that the patient thought he was rid of through his repression. This surrogate of the suppressed idea—the symptom—is secure against further attacks from the defenses of the ego, and instead of a short conflict, there originates now permanent suffering.

There are opponents of this more or less pure psychogenic point of view of the causative agent of the psychoneuroses. Golli and Mott can be quoted as outstanding authorities for this school. According to Mott, a study of a large number of family pedigrees has shown that psychoneuroses and psychoses are found in various members of a stock often endowed with genius and with high intellectuality. This fact he regarded as evidence of an inborn tendency to neural irritability affecting neuronic structures of the highest and latest evolutional levels. He was certain that a system of physical methods of measurement could be applied to the objective study of mental processes and utilized in neurologic clinical diagnosis. Golli stated that one is justified in assuming an organic disability as an antecedent to every neurosis and in employing methods for the objective evaluation of organic efficiency in looking for it. (He was speaking of the war neuroses at the time.) Further, the pathologic process of dementia praecox is recognized as a degenerative condition affecting practically every bodily function. Researches of Pighini, Mann, Mott and others have shown that profound metabolic disturbances are the functional expression of many anatomic changes. Golli stated: "Since dementia pracox is merely a unit in the constellation of hereditary psychoses any of which may equally well befall the other members of a psychopathic family, there is but little doubt that a similar organic basis will be discovered for all

the psychoses." Veeder, in a personal communication to the author, stated: "An organic basis for such conditions as dementia pracox (and hence for many allied psychoses) is far from proven. Some of Mott's patients died of tuberculosis, and the changes found in the brain might well have been due to the latter disease rather than to dementia praecox." The various opinions here are sufficient to illustrate the wide variance present with different authorities.

SIMULATION

Before considering further the general as well as the specific ocular symptomatology of hysteria, it is relevant to consider malingering briefly from the same general psychologic basis, i. e., the underlying raison d'être for its practice. To begin, there are no reasons why the malingerer should not present stigma and symptoms of hysteria, nor is it especially rare for a hysterical person deliberately to simulate symptoms. There is a similarity between the two conditions both in symptomatology and in the mental or psychologic make-up of the personality. Relative to this personality make-up, Jones and Llewellyn will be quoted frequently in the following paragraphs.

The imitative faculty is highly developed in certain animals, in savages, in young children and in the insane; in short, imitation is typical of a certain grade of mental evolution. It is not strange, therefore, that malingerers so often are weakminded, have constitutional psychopathic disorders, are degenerate, hysterical, of inadequate personality, are persons of low moral sense, deficient self-control and hypersensitive to example and to suggestion. Partly in accord with Dufestal, malingerers are divisible into three major groups: (*a*) those of normal mentality, (*b*) the degenerate malingerer and (*c*) the psychopathic malingerer.

The malingerer of normal mentality is the ordinary common-place cheat, the child who shams a headache to shirk school, the man who to evade the responsibilities of citizenship feigns illness to escape giving evidence (though one must be careful that a fear of possible consequences does not modify this), the soldier who alleges illness to avoid drill or fatigue, the workman who exaggerates the result of an industrial accident, the insured person who delays his return to work, or the person who attempts to obtain through deceit, compensation for conditions not connected with an industrial disease or an accident under investigation. Malingerers of sound mind, strange to say, often prove to be clumsy and unskilful deceivers; even laymen may penetrate their flimsy disguise. Simulation for such is irksome, consequently they lack tenacity. With them malingering is short lived, to be relinquished promptly if detection threatens or as soon as their end is attained. Their purpose is not the result of any ingrained proclivity toward

deceit, and, sensitive to shame, they either confess by affirmation or perhaps by innuendo and cease their simulation.

The degenerate malingerer is of an entirely different stamp or cast. He is most frequently encountered in prisons, in reformatories, among tramps and mendicants and among persons who seem to be of a low grade dementia praecox type. He is of "les mauvais subjects" who prefer idleness to industry, and in whom feigning of disability or of disease is but a part of his general revolt against law and order. This person contrasts markedly with the malingerer of normal mentality in his persistency, in duplicity and in the polymorphous protean resources he presents. The shrewdness shown by many of these persons is uncanny. To match one's wits against them is often a battle of no mean forces. There is no such thing with them as confession of duplicity. They go down, usually, when exposed, still fighting regulations and verdicts no matter whether these are before a military court or before an insurance referee. As Jones and Lewellyn said, "for such, malingering is a well thought-out scheme to be adhered to at all costs. Once committed to a plan or campaign, they follow it out logically, without contradicting themselves or falling into the snares set for them." They are indifferent to discomfort and tolerate without flinching any painful tests which may be used. These patients occasion the most trouble for an expert, and detection can be achieved only after skilful and often long-continued observation.

They may exhibit stigma of degeneracy, but these may not be of diagnostic import save when connected with a family history of neuropathic antecedents. Under such circumstances, the difficulty in the diagnosis is lessened. One must not make the mistake in dealing with these patients to think that this state of degeneracy is synonymous with illiteracy or with ignorance. If so, the investigator is lost at the start.

The psychopathic malingerer (hysterical, weakminded and insane) is a much more difficult problem as to his peculiar traits. He is seen in clinics and in hospital wards with diagnoses ranging from intracranial neoplasm to epilepsy, from complete amaurosis to a high degree amblyopia, and it is puzzling to know just where the neurosis stopped and pathologic fabrication began. It seems as if the pathologic fabricator properly falls into this subdivision. This diagnosis is not at all amiss in certain instances. On the other hand, the hysterical symptoms and objective changes reveal their nature. Motives also are quite evident in these cases.

As malingerers, these persons may be obstinate like the degenerates, but, unlike them, they lack constancy—an unswerving adherence to a preconceived scheme for fraud. Preeminently suggestible, they are "to one thing constant never." Their complaints and disabilities vary kaleidoscopically. As in the other malingerers, many of these symptoms are

a manifestation of voluntary deceit rather than a part of morbid neurologic symptomatology. Babinski, Strum and White, each separately refuse to consider this hysterical simulation, i. e., automutilation, suffering, etc., in any light except that of deliberate deceit. Still, these cases, while they illustrate a foregoing statement, are the bête noire of the thoughtful expert who would and must separate the feigned or exaggerated from genuine or hysterical manifestations.

The weakminded or insane malingerer is of little concern in this differential discussion. The established diagnosis of insanity, when known indelibly, brands all attempts at simulation. On the other hand, it is relevant here, if for no other reason than its academic discussion, to complete the picture of the simulator, to include those who simulate insanity, rather than the insane simulator. As Jones and Llewellyn said, "while normal persons may simulate insanity, most experts agree that the majority of examples of feigned mental aberration occur in persons, strictly speaking, of unsound minds." As Laseque once remarked, "we simulate only what we have," and doubtless this is the explanation of many cases of feigned insanity. It is not without significance that most of these cases of feigned mental disorders occur in criminals who assume this guise as a plea for irresponsibility, while others occur in persons of an epileptic type, in paranoid or paranoiac persons. Still the mental flaw in many of these patients is difficult of detection, their shrewdness and cunning so often seems incompatable with a true insanity. Consequently, complete analysis by modern psychiatric methods is a sine qua non if their measure of responsibility, or the opposite, is to be approximately gaged.

From an ophthalmologic standpoint, neither of these varieties are of great interest, the insane person who simulates or the sane person who simulates insanity. The latter is entirely foreign to the ophthalmologic domain, save for the occasional requests for determination of visual acuity, fundus examinations and perhaps determinations of the peripheral field. The former may be more commonly met with, but the fruits of the simulation, even if this is not detected, are futile from economic standpoints, except in those cases in which the insanity is undiagnosed.

It seems as if the factors of greatest importance in such cases are first that the basic psychosis is undiagnosed, and the second, which almost amounts to the same thing, that the patient is not under institutional care or under active observation and guardianship.

In a few of the foregoing paragraphs, a consideration of the mental status of the malingerer, was given with the idea of determining the type of person who will simulate. To simulate means to deceive deliberately with a more or less cold-blooded determination. This can be rather horrible at times, and at other times almost laughable in the

absurdities that develop. But just as amusing inconsistencies arise in hysteria which makes careful examination and correct interpretation necessary. For example (Jones and Llewellyn), "How bewildering is the attitude of a woman who swears she cannot feel the softest contact," in testing for epicritic and protopathic senses, "and still in spite of closed eyes says no to every isolated touch with the pencil or the finger." In such an examination the patient's eyes are bandaged, and she is expressly asked to say yes when she feels something and no when she feels nothing. Her answers conform strictly and synchronously with the contacts, the negatives as well as the affirmatives, and one is confronted with this dilemma: "She asserts that she has felt nothing, while in truth her replies show that she must have felt something."

But given the mental background discussed in such detail, that which precipitates the act is always the possibility of some definite tangible reward. The extremes to which some persons will go to achieve their end are unfathomed. During the World War, I was a member of a general court martial trying soldiers accused of self-inflicted injuries, done to escape the possibility of death or of more serious wounds than those self-inflicted. These were invariably of a less serious nature than possible through the enemy's fire, but they included grenade and gunshot wounds resulting in the loss of a hand, a perforating wound of the foot, usually the left, or of superficial stab wounds, attacks of chemical dermatitis, cases of traumatic conjunctivitis, of self-inflicted gastro-enteritis, etc. An eminent neuropsychiatrist was also a member of this same court martial, and it has often been a point of some conjecture with me, in recent years, if the presence of this member did not save many a neuropsychiatric person from military punishment which would have been rather ill deserved. In such instances, and during the high tension of modern warfare, it is often a question and one not always answered with impartial justice, whether to punish or to treat? The differential diagnosis of a pure situation neurosis and of pure malingering, for these self-inflicted injuries, was a most difficult matter. Even these extreme cases, however, were no more difficult than are many of the ophthalmologic cases presenting themselves every day in both outpatient clinic as well as in private practices.

Before further attention is paid to intensive diagnosis, it is well that several statements are explained further, if only to add necessary emphasis.

The hysterical person is largely, if not wholly, unconscious of the unreality of his symptoms, more given to extravagant and bizarre comparisons. He deceives himself, not seldom others, without design and without malice, often without demonstrable motive, although this last is in existence but buried. The demeanor and the behavior of the

hysterical subject (Llewellyn and Jones) "shows indications of his abnormal mentality. The excitability and restlessness, the hurried speech, the odd gesticulations, the rapid muscular tremors, the dilated pupils and the widely opened eyes, as if frightened, all combine in a familiar characteristic expression." The course of his disease is not uniform; it may be subject to great variations and to intermissions. The sudden disappearance of long-standing and severe hysteria is not uncommon. Short hysterical attacks disappear as abruptly and with similar weirdness. "Design is always much less obvious in the speech and behavior of the hysteric. He is much less apt to slip into inconsistencies than the knave." When he is accused of contradictions, his attitude is rather classic in its mixture of pose and passion. The ordinary deceiver is quite incapable of reproducing this. The hysterical patient revels in examination; it cannot be too long or too minute. The larger and more interested his audience, the better does he attempt to demonstrate the severity, the pain and discomfort, in fact, the unusual nature of his condition; as the woman with hysterical angina pectoris remarked to Jones, "I feel as if my chest was being torn asunder, like a ship in a butcher shop, my ribs are being forced apart by a bar of wood."

The malingerer deceives others, not himself, consciously and with intent, and always from motives of reward if successful. The malingerer, though his outward appearance may much belie him, is often sullen, obviously suspicious and ill at ease. He is conscious of the unreality of his symptoms, and while he volubly protests as bona fide his impairments, he is more careful than the hysterical subject in choosing his words so as to avoid embarrassment.

As the hysterical person loves examination, so the malingerer loathes it and will do almost anything to evade it. During its progress he is often sulky and noncooperative. If he can find anything harsh in the tests or the methods used, he is quick to take offense. It is not uncommon for him to counter with abuse. He seems to sigh with relief when a period of examination is over. However, simulators who carry on their deception with most unusual fortitude and at times with apparent indifference are not uncommon. As a rule, they are likely to put aside their rôle of deceit when they feel free from observation, for they, as is natural with every one, do not like unnecessary inconvenience or discomfort. In this attitude, the hysterical person is more consistent. Even when not knowing that he is under observation he maintains his characteristics, though prone to exaggerate them under the stimulating effect of bystanders. The hysterical person, when recovered, is often rather jubilant over the miracle; the malingerer, when uncovered in his simulation, often goes on as said before, fighting decisions and regulations to the last. As Spear said, "the malingerer seems to belong to a

paranoid class and often becomes litigant." The same applies to the person with latent hysterics. His requirement as to differential diagnosis, which is enlarged on shortly, follows. Is the production of the symptoms based on organic nature as compared to those based on a psychologic foundation? The former is the wilful desire of the malingerer, the latter the unconscious act of the hysterical person.

POSTTRAUMATIC RELATIONSHIPS

E. Fuchs, in discussing hysteria and simulation, when connected with accidents and injuries, called attention, in the following quotation, to a most important comparison and to possible developments.

> But the real test of an expert are those cases in which the function of the eyes is affected simply by shock, or the psychic commotion that accompanies the injury. These cases are known by the name of traumatic neuroses, and, so far as their essential nature is concerned, belong to hysteria; hence the disturbances of sight that are present must be characterized as hysterical amblyopia and hysterical asthenopia, produced by an injury. . . . If we find them in a workman who has been injured only a little while and has not yet had any medical examination of the eyes, we shall be able to make the above diagnosis. But it is otherwise when we have to do with patients, especially those who have been through a railroad accident, who make claims for big damages and have been already examined a number of times, particularly if they belong to the cultivated classes. Then, there is the danger that the symptoms have been suggested to the patients by the repeated examinations or that they have become more and more conversant with the symptoms, about which they have been questioned and then intentionally simulate them. Since in default of objective findings we are altogether dependent on the patient's statements, it is in many instances absolutely impossible to detect a clever simulant.

During the recent war these cases assumed a marked importance from a medical as well as a layman's standpoint, the latter being fostered to a great extent by the press. The condition has been known for many years, especially through mine disasters in which explosions occurred within the relatively small contained spaces of mine cuttings. The misnomer, "shell shock" was changed as soon as was possible, but it is still too well known by this name, though incorrect. The eye syndrome of a postconcussion neurosis state, without any definite gross organic changes intercranially, is as follows: some inequality of the pupils, not uncommonly unilateral impairment of pupillary reflexes even to their complete abolishment and an irregular concentric contraction of the visual fields. The amblyopia found in these cases is unlike that of hysteria. It is more commonly an amaurosis rather than an amblyopia, or with massive central scotomas. Fatigue fields are also not unusual later in the course of the condition. In these cases the first and most important point is to determine whether or not organic lesions of the brain, i. e., the optic nerves, the chiasm, the nuclei or the occipital lobes,

are present. As previously stated, the pupillary reflexes and the visual fields both in peripheral as well as central field defects, are of primary importance. When once the cases are sorted into organic and non-organic classes, entirely different treatment is given; the former are purely surgical cases, and the latter, without any doubt, are medical cases. The patients in the latter types give one the opinion that if they see they do not know that they see. Llewellyn and Jones stated:

> If, in examining the eyes of patients suffering from concussion, amblyopia, the light reflex, even when no sight is acknowledged, is present, we argue therefrom that the primary optic centers, and all that they connote, are not involved. The patient avoids obstacles: he does not run against them, taking care not to hurt himself, as the malingerer does, but he hesitatingly goes around them. It would seem that the impression has reached his occipital lobes, for the information is acted upon. At least, it has reached his reflex instinctive "center," but it is not recognized because it has escaped his psychic ideational "center"—a very simple explanation—but one requiring the postulation of praxic centers for psychic vision, hence also (in terms of hysterical manifestations) for psychic audition, and for psychic vocalization, centers which have supreme control over the highly organized functions and manifestations of life. The hypothesis of an orientation center distinct from that of vision may account for the psychogenic phenomena of pseudovisual impressions. Such a sense would annul the necessity of sight in the cases we are considering and would explain how it is that the patients avoid obstacles without "seeing" them. But in this as in other speculations it is not advisable to lay too much stress upon the imagination as a basis for theories.

Eszenyi's explanation of this spatial orientation in the peripheral portion of a tubular field can thus explain the ability a patient had to play a brilliant game of tennis in spite of tubular fields.

This condition of amaurosis or severe amblyopia may go on for weeks as a complete dissociation between the patient and the outside world. Suddenly, and at times without the prodromes found at its onset, the vision returns in its entirety. The patient's mental shock of meeting his family, his wife, that of a severe lightening and thunder storm, etc., may be sufficient to tear aside this curtain between his retinal perception and cerebral vision, the reestablishment of continuity with the psychic ideational center previously mentioned. Occasionally, the recovery is not as dramatic as this; instead, the patient recovers slowly.

A certain number of these patients with purely functional conditions recover a large percentage of central vision but still complain of foggy or hazy vision. Such patients, when examined further, will fall within the class of psychoneurotic neurasthenia. The fields are irregularly contracted, with irregular, nonsystematic defects. Occasionally the color fields are found disturbed, while the form fields remain practically normal. At other times, the defect lies wholly in the peripheral fields. This type of case is illustrated by the fatigue fields explained by Peter in his "Principles and Practice of Perimetry."

DIFFERENTIATION

Both hysteria and simulation are definite pathologic entities. Each case that is presented for diagnosis should be classified into one or the other type, but unfortunately this is commonly impossible. The reasons are not numerous: When one considers the protean manifestations of hysteria, almost the entire science of semeiology is covered. One might paraphrase Osler and say, "he who knows hysteria knows medicine," an exaggeration but still applicable. The same applies to simulation, for here the symptomatology is limited only by the fertility of the person's mind. On the basis of the works of various authors, the differential diagnosis must consider three subsections: (*a*) symptoms and signs in favor of organic disease; (*b*) a possible diagnosis of hysteria in the case under consideration and a possible combination of hysteria and organic disease, and (*c*) a grouping of contrasts between the psychic anomalies of hysteria and of malingering, i. e., is a pathologic liar (the case under consideration) one or the other? The possibility of both conditions being present in the same person should be considered. This grouping will be referred to again, but it is mentioned here first because of its relevancy.

The hysterical patient may simulate, and the simulator occasionally does develop hysteria, even to hysteria major (as has been seen by the author), to such a degree as to make sanitarium incarceration obligatory. Third, every effort must be made to dispose of each case presented, i. e., not to allow a patient to drift away into economic desocialization. Both the hysterical patient and the simulator need treatment, and that for malingering is not always purely disciplinary. The last point applies to a differentiation procedure which too often is the basis on which one works, and it might be added, on which one must work. Trapping a patient with amblyopia into seeing, a paralyzed arm into motion or an epileptiform seizure into relaxation is proof of neither one. Pitfalls are too easily laid by any one and evaded with great difficulty by the person who is under observation. For instance, in hysterical patients, according to Chavigny, cutaneous anesthesia may be regarded as a type of localized mental distraction. Trickery and acute and unexpected stimuli may suddenly arouse attention and therewith sensation. Still it would be wholly erroneous to translate such reactions as a proof of simulation.

The true diagnosis cannot be decided as easily as this except in a few cases. It depends almost as much on the failure to find certain signs, symptoms, stigmas and other classic characteristics as to find inconsistencies, absurdities and other allied factors pointing toward simulation. It is a no less regrettable error to fail in exposing simulation as to punish unjustly an unfortunate psychoneurotic patient. As in law, a patient must be proved in *flagrante delicto* before simulation can

be claimed; if proof is not established by absolute confession then it can be arrived at only through conclusive evidence. There can be no question of doubt. The familiar tenant that "mistakes are easily forgotten when publicly unknown" is as dangerous here as in any other circumstance.

THE SYMPTOMATOLOGY OF HYSTERIA

The mimicry of hysteria is misleading, according to Head, and only apparent to the inaccurate observer or to one content with a perfunctory examination. For the diagnosis of hysteria it is not only necessary that there should be no demonstrable organic cause for the symptoms but positive signs of hysteria must be present. Hysteria as a conversion neurosis simply signifies that the conflict in the patient's mind is solved by some aberrant reaction. It removes him from a position of doubt. Without considering any of the ocular symptoms, the following is a brief résumé of the possible general symptoms:

1. Disorders of movements: various tremors of a nonrhythmic type which disappear when the patient can be persuaded to perform some other movement; incongruity of symptoms as sphincter paralysis with paraplegia or hemianesthesia without hemiplegia.

2. Disorders of speech: speech may show hysterical stutter.

3. Hysterical spasms: These depend often on various postures; an attempt to break them is met with increased resistance, regardless of the direction in which movement is tried.

4. Deviation of the tongue: The tongue protrudes and is withdrawn always deviated to the same side instead of the type present when deviation is based on an organic cause.

5. Incoordination of movement: This symptom, when present from organic disease, is due to a lack of recognition of the position (spatial) of the afflicted part. A cerebral lesion makes an impossibility of touching the nose, and as the position of the moving hand is unknown it will be even more difficult for the unaffected hand to find and to touch the affected hand. The exact opposite is true with the hysterical person. Here the unaffected side will have no difficulty in touching the affected side.

6. Anesthesia, paresthesia, hyperesthesia all without any relationship to the cutaneous nerve supply: The glove anesthesia shows one how the patient's attention is focused on the member as an entity without regard to the nerve distribution. They are of segmentary distribution. There are vasomotor derangements.

7. Hysterogenetic zones: When these zones are stimulated they show plainly that it is the position and the presence of a painful zone and not the painful or painless nature (protopathic or epicritic) of the stimulus which dominates the consciousness.

8. Hysterical globus, hemicrania, vomiting, anorexia and mutism.

9. Convulsions, minor and major hysteria, emotionalism, exaggeration of reflex excitability and loss of will power.

10. Paroxysmal nature of the phenomena: variability, sudden appearance and disappearance of the symptoms, incongruity of the symptoms, disproportion

between subjective complaints and objective manifestations, responsiveness of symptoms to outside influences or even to autosuggestion.

11. The syndrome of pain: This is purely subjective, and when due to organic cause and continued for any length of time, is accompanied by a loss of weight, an anxious expression (Spear), a desire to be left alone, an unwillingness to discuss the pain. Relief is obtained by analgesics. When a painful part is moved there is an increase in pulse rate, sweating and dilation of the pupils. With the hysterical subject, pain is out of proportion to the causative agent; it is not relieved by analgesics, or if relieved, a placebo is quite as effective; it does not cause loss of weight or anxiety, and it does not conform in distribution, character or severity to the possible causative agent.

If any of the following changes also (briefly and incompletely abstracted) are present, the diagnosis of hysteria is doubtful:

1. Optic nerve atrophy, internal or external ophthalmoplegia, strabismus, persistent central scotomas, systematic hemianopia and true nystagmus, degenerative atrophies, paralyses of muscles supplied by a single nerve as the facial, or by a spinal plexus as the brachial, the presence of changes in the knee reflexes, the presence of an ankle clonus, a Babinski or Oppenheim's sign, Horner's or Strumpell's sign, a positive Kernig sign, or the incontinence of urine or of feces.

2. In the sensory sphere, anesthesia of any area limited to the distribution of a particular nerve trunk as the musculospiral (N. radialis, B. N. A.), or limited, as in the hand to a pure median or ulnar paralysis.

3. Trophic changes of great severity as decubitis. (Certain trophic changes of hysterical postures and with the hysterical plegias are common.)

4. In the psychic sphere, dementia, organized hallucinations, memory of the delusions and hallucinations. The classic multiple personalities are an outstanding mental stigma of hysteria.

When one considers that the following diagnoses, all proved as hysterical, have been culled from the literature, from hospital charts and from my own cases, one must consider seriously the inventiveness of human personalities. The list given is quite incomplete:

Extrasystole arrhythmia, angina pectoris, Pott's disease, tuberculous hip disease, scoliosis, bilateral symmetrical paralysis of the eye muscles, ptosis of the upper lid, tumor of the brain, multiple sclerosis, epidemic encephalitis, meningitis, laryngeal hysteria, hysterical hiccup, cataleptic seizures, hysterical deafness, hysterical blindness, hysterical monoplegia, absolute anorexia, enteroptosis, gastro-enteritis, pregnancy (pseudopregnancy) multiple personality.

It is relevant here as well to refer to those interesting hysterical stigmas of crucifixation and of the Crown of Thorns, as in the case of the Maid of Konnersreuth, as well as some of the miraculous healings which occur at famous shrines.

A brief survey of neurasthenia as compared with hysteria is included. According to Reynolds, the fundamental difference lies in that the former is principally a matter of objective physical signs while the latter is largely one of subjective symptoms. The manifestations of

hysteria as blindness, deafness, mutism, anesthesia, loss of power, vomiting, etc., can be observed and tested by others; those of neurasthenia all relate to altered feelings. It seems as if the neurasthenic person cannot recover from the fatigue incident to normal mental and physical activity, hence the nerve cells become at first more irritable and more susceptible to further fatigue; finally their function is lowered. There is no common standard for fatigue, a point beyond which fatigue must not go if ordinary repose is to restore the used cell to normal. The difference seems to lie in a necessary neurotic basis, whether it is heredity or through trauma, etc. It is a close second, however, in its economic loss to the patient and to the community when compared with hysteria.

The ocular manifestations of hysteria are not extensive as compared to its other symptoms. Head's outline is given as an index for this section.

1. Disturbances of sensibility such as anesthesia and hyperesthesia of the skin, cornea and the conjunctiva; photophobia; neuralgia
2. Visual disturbances
   - Diplopia, monocular and binocular
   - Polyopia
   - Erythropsia
   - Micropsia and macropsia
   - Amblyopia and amaurosis, one or both eyes
   - Reduced retinal function of color sense and of light sense, as hemeralopia (Landolt)
   - Disturbances in the fields of vision
3. Motility
   - Ptosis
   - Blepharospasm
   - Strabismus
   - Myopia and astigmatism through spasm of accommodation
   - Miosis and mydriasis; hysterical cycloplegia
   - Asthenopia, accommodative
   - Nystagmus; conjugate deviation
   - Insufficiency of internal recti; external ophthalmoplegia
4. Secretion; epiphora and lacrimation; decreased secretion.
5. Ocular cause (patient's statement) for insomnia, dizziness, vertigo, inability to concentrate.

A similar outline of the manifestations of ocular malingering follows. The points of similarity and the differences in the semeiology of the two are thus rather evident. McAuley's outline of ocular simulation will be in part the basis for this index. It is an outline of the most common complaints. The variability of these depends only on the fertility of the patient's mind and his opportunities for observation as to those symptoms that can be simulated.

1. Visual disturbances
   - Diplopia, binocular, monocular
   - Total blindness, bilateral, monolateral
   - Impaired vision
     - An exaggeration of a visual defect which is evident on examination
     - The discovery of an amblyopia (as amblyopia ex anopia) and the claim that it is connected with injury, industry, etc.
     - Simulation of impaired vision without organic or functional basis for actual decrease in visual acuity
2. Motility
   - Paralysis of eye muscles; inability to use the eyes in their normal rotations; ectropion
   - Spasm of lids (blepharospasm)
3. Function defects claimed; inability to use eyes because of:
   - Weakness
   - Photophobia; epiphora and lacrimation
   - Dizziness
   - Dazzling
   - Scotomas and other defects in the visual fields
   - Contraction of color fields; color blindness
4. Traumatic conditions self-inflicted
   - Traumatic conjunctivitis; keratitis; scleritis; marginal blepharitis; self-inflicted injuries of the lids; iris disturbances through the use of miotics and mydriatics and when connected with organic diseases
   - Paralysis of accommodation
5. False claims connected with certain organic diseases and their ocular manifestations in an attempt to show a relationship to injury and to industrial exposures as syphilitic endarteritis, optic atrophy and interstitial keratitis and alleged trauma, pseudoneuritis and trauma, amblyopia exanopia and trauma, high myopia and trauma, etc.

DIFFERENTIATION OF HYSTERIA AND OCULAR MALINGERING FROM OCULAR SYMPTOMS

In a differential diagnosis from ocular symptoms, the following factors must be considered. The outline follows closely that suggested by de Schweinitz.

- Sensation, corneal and cutaneous
  - Lesions of the skin and conjunctivae
- Vision
  - Central and paracentral
  - Visual fields
- Oculomotor disturbances
  - Strabismus
  - Diplopia or polyopia
  - Nystagmus
  - Ptosis
  - Blepharospasm
- Mydriasis, miosis, cycloplegia
- Light and color sense
- Asthenopia, vertigo, dizziness

Disturbances of sensation are common in hysteria. Their simulation also is not uncommon. In hysteria the bulbar conjunctiva is least sensitive, then the palpebral conjunctiva, and most sensitive is the cornea; according to de Schweinitz (quoting Gilles de la Tourette), "this is usually incomplete—the center of the cornea retaining its sensibility." The palpebral and lacrimal reflexes may both be retained, so mistakes must not be made in misinterpreting their presence or absence. The retinal lid-closure reflex may assist in determining visual acuity but not that of conjunctival or corneal sensation if the testing cotton is seen by the patinet. Spiller stated that the lacrimal reflex is retained even in the presence of corneal anesthesia. Hyperesthesia may be present in a few cases. The malingerer seldom shows corneal or conjunctival anesthesia—the contrary is usually the case and often blepharospasm and copious lacrimation are found. There is usually an unwillingness for all types of simulators (except the degenerates) to stand much physical distress or pain. Those patients presenting corneal anesthesia are unsystematic otherwise and will have none of the allied conditions usually present as cutaneous anesthesia, orbicularis spasm and the periorbital hysterogenetic zones. The latter should not coincide with the exit points of the infra-orbital and supra-orbital nerves. Simulators are not alone in deliberately causing traumatic conditions of the conjunctivae. De Schweinitz described one patient who purposely scratched her corneae with pins.

Chaillous described a case of feigned petrifying conjunctivitis. Hollenberg described a case of miliary herpes, confluens necroticans—a psycogenic dermatitis—in hysteria. I have seen two similar cases of miliary pustular condition of the skin of the lids and the conjunctivae in simulators, both self-inflicted; in one the causative agent was unknown and in the other it was oil of mustard applied to the skin. Chewing tobacco conjunctivitis is not uncommon, and Lindner, in a personal communication some time ago, mentioned a deliberate attempt at cross-infection of trachoma which occurred in the military service. Vasomotor disturbances of the conjunctivae are rare, but occasionally found. Loddoni has discussed in great detail conjunctival and subconjunctival hemorrhage in hysteria; de Schweinitz and Spiller both have referred to the description of Hensinger and others relative to this.

In discussing the loss of visual acuity, amaurosis rather than amblyopia is first to be considered. It is present in both conditions, bilateral and unilateral. Naturally, it is necessary to eliminate any organic cause for the complaint. This search must include the probabilities of congenital as well as of acquired ocular, cerebrospinal and other general systemic disease. As Villard has outlined, a sudden onset, a short duration and a previous history of blindness are almost

pathognomonic of hysterical amaurosis. Still, cases of several years' duration have been accepted as hysterical. De Schweinitz mentioned a case of Harlan's of ten years' duration, and Esteban one of seven years' duration in which recovery had not yet occurred. I have had one of over two years' duration. A lengthy duration is decidedly against malingering. In this the onset should also be sudden. Intermittent attacks of blindness are seldom from simulation; they are usually functional. As Janet and McAuley have outlined, in this condition a study of the pupillary reflexes is of great importance. These are absent in true blindness (McAuley), except (*a*) when the lesion is high up in the optic pathways above the fibers of the reflex arc to the center for pupillary movements, (*b*) in bilateral lesions of the occipital cortex, (*c*) in temporary retainment in uremia with amaurosis, (*d*) in blindness from certain diseases of the optic nerve wherein it seems that the pupillary fibers for the light reflex were more resistant than the visual fibers and (*e*) in that reaction described by Leber with certain forms of retinal degeneration wherein a slow reaction to light remains even with a total absence of light perception. If these can be eliminated, one is probably justified in placing the amaurosis in the class of one or the other.

The hysterical person retains, however, a subconscious perception, (Jolly and Eszenyi separately) which saves him in many unusual instances. They do not behave like people really blind, will escape obstacles, dodge moving objects, and often they also complain of flashes of light, of photophobia, of erythropsia and other impossible chromatopsias. As Jolly said, they are a perplexing problem. When talking with these patients one is always impressed by their desire for sympathy, by their willingness for examination and by their apparent acceptance of the amaurosis—not with anxiety or fear for the future, but with a rather inconsistent nonchalant or indifferent attitude. This is not so with the malingerer or with the person who is genuinely blinded. The malingerer seldom simulates bilateral blindness. When he does, he is "blinder than the blind." He can or will do nothing for himself; examination is usually difficult, and he often has accompanying symptoms of traumatic conjunctivitis or keratitis.

Unilateral simulation is more common, as is unilateral hysterical amaurosis, apparently, less common. (See de Schweinitz for an explanation.) In the unilateral types differential diagnosis is always difficult. Herein the various trick tests for binocular vision play a large rôle. It must first be proved that there is vision in the alleged amaurotic eye before differentiation can be considered. Following this one may cast up a balance sheet of negative and positive symptoms for the decision. Relative to the diagnosis of a true unilateral blindness (McAuley), it is well to remember that a blind eye does not share in the

process of fixation, but moves into a position of anatomic rest. As Janet stated, after vision has been proved in the amaurotic eye, the medical examination reveals the hysteria. It is also relevant here to recall that most unilateral hysterical amaurosis is connected with emotional excitement, with various painful mental episodes, with certain physiologic states, and is quite commonly recurrent. The investigation of the field of vision in the opposite eye may demonstrate hysterical contracture.

The amblyopia of hysteria is described often as painful vision, as vision with spots and with colored lights. Asthenopia accompanies it frequently. An amaurosis in one eye with an amblyopia in the opposite eye is rather likely hysterical though this also is the forte of the simulator. Disturbances of binocular vision, the dissociation of Antonelli (de Schweinitz) and of Balduzzi, especially with an unequal bilateral amblyopia, is likely to be hysterical. There is also in this an occasional alteration of the degree of amblyopia. In hysteria it is uncommon to obtain through various trick tests varying degrees of visual acuity, usually in an ascending scale of increasing distance vision. This is classic when examining the malingerer. It has seemed to me that the malingerer with an alleged impaired vision is capable of doing more with this damaged vision than the hysterical person or even the patient with a true bilateral amblyopia. He (the simulator) seems unable to gage acurately probabilities of achievements consistent with the amblyopia.

In hysteria, perimetry assumes the proportions of an outstanding characteristic. In malingering, this remains decidedly secondary; hence, one might say, from the standpoint of differential diagnosis, perimetry plays a most important rôle. According to Eszenyi, these authors, Strumpell, Oppenheim, Jendrassik, Bausch and Heilbrommer, claim that changes in the fields are not diagnostic. Bing, Kraepelin, Moraycsik, Charcot, Janet, Schaffer and many others insist that if not of specific diagnostic import they are most valuable. Of 300 studies of the visual field reported by Eszenyi on psychopathic patients, 200 were in cases of hysteria. Of these, only 3 were normal. One hundred and ninety-seven patients showed various forms of contractions, color inversions and dyschromatopsia. Sixty-six of them showed color inversions. Of the remaining 100 patients, 50 with schizophrenia had 17 normal fields; 10 with multiple sclerosis had 6 normal fields; ten with unclassified cases of paraphrenia had 9 normal fields; 20 with jacksonian epilepsy showed first or second degree contracture and inversion. He also found, as did Parinaud, Charcot, Landolt, Lazenbeck and Mehring, that in hysteria the color amblyopia develops in the following order—violet, green, blue, and last, red. This when compared with the red-green loss of optic nerve atrophy is most interesting.

As a result of his observations, Eszenyi concluded that hysterical field changes are of diagnostic importance in differential diagnosis. This is also the same point of view of Peter and of Ronne. Further, he stated that the dyschromatopsia can be explained through the Young-Helmholtz-Hering theories by hypo- and hyperesthesis of the retinal elements. He further added that maintenance of visual orientation in that part of the peripheral retina represented in the field through its hysterical contraction as blind is a pure spatial automatic reflex. His last stated contention is that the field changes illustrate or allow one to consider a disease symbiosis of hysteria with other psychoneuroses and psychopathologic states as neurasthenia and hysteria; multiple sclerosis and hysteria, and hysteria with morphinism. Quite properly the author adds here simulation (malingering) with hysteria.

While relevant in this review only with bare mention various authors have described other peculiar relationships as they exist clinically, i. e., hysteria with multiple sclerosis, with encephalitis (chronic), with syringomyelia, with epilepsy, with exophthalmic goiter, with pseudobulbar disease, with chorea, with migraine, with certain forms of insanity and with other conditions less common.

Rakonitz drew attention to the following factors which without doubt play a part in the field changes of both hysteria and malingering though the method by which they act is different: (*a*) faulty and disturbed attention, (*b*) autosuggestion, simulation, psychic impressions and impulses, (*c*) a claim or acknowledgement of amblyopia, either psychic or otherwise, (*d*) a narrowing of the conscious, that is, the imprint of the preconscious and the unconscious, (*e*) changes in the irritability of the visual projection and (*f*) perhaps certain other unknown components.

Bing laid great stress on these field changes. Binswanger stated that the study of the fields is of greatest value. Babinsky said that while hysteria and hysterical manifestations have changed from age to age (as mankind and his environments have enabled him to change the symptomatology), the hysterical contraction of the fields of vision has remained unchanged throughout these years. Charcot stated that field changes are pathognomonic of the condition. Gillies de la Tourette considered them as one of the most outstanding of the hysterical stigmas. Wilbrand, Saenger and Parinaud mentioned the analogy between contracted fields and cutaneous anesthesia. In fact, they saw the former most commonly in cases with this latter stigma. Peter, from his personal observations, stated that the possibilities of field changes in hysteria are, with a few exceptions, limited only by the patient's knowledge—a simulation not of malingering but of inhibition. He, with several others, Förster, von Reuss, Wilbrand and de Schweinitz, stated that the oscillation and spiral fields should be classified with those of hysteria.

Many men have seen a similar symptomatology of changes in the visual fields in malingering, almost as characteristic as with the aforementioned. Rakonitz and Hitzig have been quoted relative to this, and Llewellyn and Jones have also called attention to the fact. Ziehen, Lorenzetti and Klein have all found such suggestive changes in fatigue, in simulation, in arteriosclerosis cerebri, in commotio cerebri, in dementia senilis, in depressive states and in other allied conditions. Binswinger and Nonne have warned against forgetting the diagnostic importance that certain field changes might have in hysteria, but with Thomsen and Oppenheim they stated that they are not purely characteristic of hysteria, for simulation, epilepsy, alcholism and certain metalic poisons all have fields of great similarity. I have found some types of field changes to be an almost constant symptom in ocular malingering, and the differentiation of these from the changes of hysteria is a difficult problem in certain instances.

The various changes which occur are almost legion. They range from the well known concentric contraction through tubular fields to the frank homonymous hemianopia of the higher cortical type found by Janet. Goldstein considered the concentric round contraction the classic characteristic. Bleuler considered the tubular field as classic. Klein stated that a field is only tubular when it not only remains of the same extent with recession of the test object from fixation but actually may become smaller. Hitzig, on the other hand, considered a tubular field characteristic of frank simulation far more frequently than with hysteria. Rakonitz gave his experiences as follows: The concentric contraction for form and color is irregular; there can be overlapping or total inversion of the red and blue fields; the blue field is in greatest contraction, thus it can overlie the red. These changes incident to hysteria are almost always bilateral, form changes and color inversion (Verschüebungstypen); fields are larger when the test object is moved toward the point of fixation, "zentripetaler," than when moved from the point of fixation, "zentrifugaler"; and the extreme rarity of central scotomas. He considered the following changes in the fields not constant: the shape of the field may be round or elipsoidal more nearly resembling the normal shape and with the retention of temporal projections; the outline of the color fields may be irregular, jagged, or they may lie in smooth bow shapes; the presence of tubular fields, and rarely a field of apparently overdimensions for both form and colors. De Schweinitz has also mentioned the latter.

If one recalls the psychoneurotic element in all types of malingerers, and refers to the quotations just given from Eszenyi, Oppenheim, Rakonitz, Lorenzetti and others, it is not at all strange that these must be studied in all cases in which malingering is suspected. The shifting fields mentioned, the fields of inversion, those changes of dyschroman-

opsia, tubular fields, hemianopias, the rare central, and paracentral scotomas, annular and wedge-shaped scotomas, all are as probable in the one as in the other. Blind spot changes in fields of sufficient extent were, it seemed, more often present in malingering than in hysteria but there are other circumstances which explain this in part. It seems as if Peter's statement relative to hysteria is as applicable to malingering, "the different changes possible are only limited by the knowledge (and ingenuity) of the patient." Repeated examinations are far more important in the latter, however, than in the former. I recall the case of a man subsequently tried before a medicomilitary board wherein two field examinations were done daily for a period of seven days, four different forms of apparatus being used, with about the same results, the evidence of a superior altitudinal bilateral irregular hemianopia, without regard to the physiologic modifications in the field that a genuine pathologic process would have demanded. The patient recovered spontaneously and confessed deliberate simulation following a severe bout of middle ear disease. Until that time a positive diagnosis was impossible.

Changes in the visual field in simulation are not found in the almost 100 per cent of instances by most men as was found by Eszenyi. Suggestion will develop changes in many repeated perimetric studies wherein any form of suggestion whatsoever (if this is possible) was rigidly eschewed in the earlier examinations. But this is not peculiar to either condition.

A philosophic discussion of that which underlies hysterical amaurosis, amblyopia, light-sense and dyschromatopsis is not relevant herein. Peter quoted de Schweinitz's work on hysteria in which Binet and Janet alleged "that patients suffering from hysterical amaurosis, when placed under hypnotic influence, can recall what they saw before the hypnotic state was induced during the amaurotic period." Doubtless the temperament of a malingerer would make this test difficult, but if it could be used it would be interesting, and perhaps valuable as well, to know its outcome. It is well to say, however, that these factors are undoubtedly not present in purely deliberate simulation. If a case is approached with this in mind, it seems as if differentiation should be less difficult.

Disturbances such as strabismus, diplopia, nystagmus and ptosis, within the confines of this review are almost wholly hysterical. Balduzzi discussed his case of hysterical contracture of the internal recti correctly from this standpoint alone. De Lapersonne and Cantonnet have the same point of view. Parinaud, Schweiger, Borel and D'Hilot have all covered this symptom rather well. According to these authors, the strabismus is of various bizarre types, esotropic and hypertropic, some alternating, and some monolateral in character, and with or without mydriasis, nystagmus, ptosis, amblyopia and amaurosis. The important

factor is that these cases of strabismus are never paralytic in either condition. Underlying weaknesses undoubtedly play a part as pointed out by several authors—convergence—divergence relationships, thus showing a deviation as the result of a convergence spasm plus a divergence weakness. The opposite would be true as well. In these cases certain characteristics seem to occur. The relationship is upset between muscle paralysis and lateral movements, between voluntary movements and "automaticoreflexes" (Parinaud, Ballet, Raymond and Koenig and others quoted by de Lapersonne and Cantonnet), the nystagmoid movements of extreme lateral rotations in pareses and in the vagrancies of the diplopia. The malingerer would be certain to disclose the simulation.

Isolated paralyses of a single ocular muscle have been described, but these cases differ from the general class of convergence-spasm divergence-insufficiency (or the opposite) cases only in that they represent more likely instances of associated movement impairment discussed below under conjugate deviation.

The usual attempt of the simulator is to allege a traumatic or industrial connection with the preexisting nystagmus, strabismus or ptosis under question. Hence there is no problem of differential diagnosis between hysteria and simulation. The case is to be decided from another standpoint wholly.

A complaint of diplopia is somewhat different. Various dissociation tests are necessary. Absurdities will always arise. Prisms and a stereoscope will be needed. Binocular vision must be determined. The loss of this is rather often hysterical. Also, this resulting monolateral suppression may alternate. Suppression and amaurosis are seen together frequently. The absurdities which arise are readily discovered. Those which are disclosed as most consistent are hysterical according to Ulrich. The alleged diplopia of simulation can be changed by the examiner as often as he wishes. Physiologic diplopia may present itself at times as in a case which I saw with Peter. Some difficulty may arise to determine the reason for the patient's alleged disability in such a case. Undoubtedly a strong psychoneurotic element must be present in such cases.

The diplopia of hysteria is not necessarily dependent on strabismus. It is not uncommonly monocular, as in simulation, in fact it is usually monocular except when the result of hysterical strabismus. With this it is an atypical type of diplopia rather similar to that of true divergence paralysis. (See convergence spasm with divergence weakness already quoted.)

The witlessness, however, of persistent uncrossed diplopia in a divergent strabismus, with unilateral high myopia and with amblyopia alleged to be the result of a situation neurosis is rather evident. (Case of Capt. G. B. H. Terry's, unpublished.) Many similar cases, however, will arise.

Hysterical ptosis is usually spasmodic (de Lapersonne) and is frequently accompanied by a quivering blepharospasm during the examination. The ptosis of simulation must always be purely spasmodic and involving the upper lid. The involvement of the lower lid is usually much less evident. Relaxation of the ptosis is not necessarily a sign of simulation. The absence of a complete obicularis blepharospasm is more important. While in this review, ptosis and blepharospasm are the same thing, the amount of obicularis contraction (upper lid alone or both lids) seems to be an important and distinguishing factor. Corneal anesthesia at the same time is not uncommon when hysterical. Spastic mydriasis is occasionally present, though miosis is perhaps more common. According to Babinski, Sauvineau and others, hysterical mydriasis and miosis are always spastic. They deny the existence of an hysterical internal ophthalmoplegia. The presence of the photomotor reflexes (Pansier, de Lapersonne, Benoist) will determine this. An upward rotation of both eyes during forced elevation of the lid is rather common with the conscious contraction effort of the orbicularis necessary in simulation.

It is almost impossible to open the lids of a case with hysterical ptosis because of the blepharospasm. The patient with hysterical ptosis occasionally complains of it being painful. In these the blepharospasm is marked, photophobia is at times present and the cornea, conjunctiva and skin of the lids is hyperesthetic rather than anesthetic, though anesthesia has been described. The author has never seen this combination of painful ptosis, hyperesthesia, blepharospasm and photophobia in simulation. The simulator may occasionally complain of ptosis as the result of pain and photophobia, but not as a complication of it. There is a difference. The simulator with ptosis allows lid opening even to a wide degree by simply continuing a slight upward traction with the examining finger for several minutes. Fatigue sets in soon. A lid elevator is not as satisfactory. The orbicularis contraction of the hysterical persons seems to increase instead of to decrease as an attempt to open is persisted in. Flaccid ptosis, hysterical, from a levator involvement, is without doubt rare. Its possibility is not inconsistent with other hysterical manifestations. Menestrina doubts its presence. Déjerine thinks it might occur in hysteria. Bernheimer and Babinski deny its presence, and the latter bases his opinion on the behavior of hysteria in the field of peripheral nerve reactions. De Schweinitz, however, has covered this controversy fully.

It is sufficient to state, for simulation, if a flaccid paralysis of the lid is present, it is either genuinely connected with the injury or disease alleged, or it existed prior to this and is only linked up with it by an error or by deliberate deceit on the part of the patient.

The question of hysterical nystagmus is controversial. De Schweinitz and de Lapersonne quoted Sabrazès (also Cabannes) as having seen it;

Borel has discussed it in detail and insists on its presence; Charcot used it as a point in differential diagnosis between insular sclerosis and hysteria (hence disclaimed its presence); Santos Fernández reported its presence, and Raymond and Souques in turn denied it. As de Schweinitz pointed out, the discussion is relative to nystagmus and not to the "rapid and sometimes bizarre movements which occur during hysterical attacks." Wolffberg described a case of right amaurosis with a left achromatopsia, with spasmodic convergent strabismus and ptosis and with nystagmus. The same relationship exists with nystagmus and with simulation. Its presence suggests either organic disease or an industrial relationship (not to forget the nystagmoid movements—ocular nystagmus—seen when fixation was not present or is lost), and it must be carefully investigated.

Blepharospasm is common to both states under investigation. It has been covered under ptosis for hysterical pseudoptosis, a word coined by Charcot and Parinaud, is essentially blepharospasm.

Conjugate deviation when not of organic central nature is a purely spasmodic contraction of associated movements, usually of a parallel nature, and when present can only be hysterical and never within the possibilities of the simulator; hence the differentiation lies only in the eliminating organic pathologic process of the central nervous system.

Miosis, mydriasis, cycloplegia and cyclospasm, when hysterical, occur in either a spasm of the ciliary body as in pseudomyopia, or in paralysis of the ciliary body as in the occasional low degree hyperopia seen in cases in which hysterical accommodative asthenopia is suspected. Hysterical miosis is rare; in fact, it is not mentioned by many authors. Those who do discuss it agree that it is spastic. (See, however, hysterical miosis when it is a complication of hysterical ptosis.) Mydriasis is common, and as stated before, the preponderance of opinion seems to indicate that it also is essentially a spastic contraction of the dilator fibers. The relationship between monocular polyopia, pseudomyopia (transient myopia of cyclospasm), mydriasis with light contraction reflex intact and convergence spasm is a rather consistent symptom-complex. Paralytic mydriasis, however, has been described by many authorities as quoted by de Schweinitz, Bing and Parinaud. It may or may not be connected with the hysterical paralysis of accommodation just mentioned.

These same conditions as related to malingering can occur in simulation only through the deliberate use of miotics, of mydriatics and of cycloplegics. I have had one case of simulation with amblyopia, corneal anesthesia and with paralytic mydriasis and cycloplegia. The patient refused to accept his retinoscopic measurement to improve vision. In addition, the patient had the diagnosis of a constitutional psychopathic condition at the time of his admission. It was never discovered from

where the patient obtained the necessary drugs for his deception, nor the time and method of their instillation, but a plaster of paris occlusive head bandage resulted in complete recovery.

The light and color senses are affected in both conditions. I agree with several of the writers relative to this. It was so demonstrated with both the Birch-Hirschfeld photometer as well as with his own light sense meter. The usual color sense defects go pari passu with those of the other field changes, and they have been referred to before; so also the cause of the light sense defect. Inconsistencies in this, as pointed out by de Schweinitz in quoting Binswanger, should make one suspicious of malingering. This is quite possible but is no more conclusive than some other allied observations. One patient seen recently at the Graduate Hospital of the University of Pennsylvania had a definite light minimum defect to the five-point diaphragm test but a much more nearly normal sense with the wedge scale. Dark adaptation developed with him readily and when full was followed by a slow return to full light adaptation. It was a full one-half hour before he could distinguish blue from black and red and green were confused for about the same length of time. It simply happened that these contrasts were tried first after recovery (when correctly distinguished), the blue-black then being shown immediately after the red-green. He had had a similar attack of complete hysterical amaurosis before, and when seen during the second attack described the former rather well. The light sense test was not done until the patient had made a spontaneous recovery from the amaurosis.

If, however, one finds a normal light sense with abnormal fields of vision, or a defective light sense with normal fields (a balanced relationship exists between the color sense, the light sense, and the peripheral fields), one may be suspicious of malingering. Color blindness is occasionally alleged by men to escape military duty of certain kinds. This could hardly be of a hysterical nature. It must be decided that congenital daltonism is not present. Certain yarn tests, a color lantern and the spectroscope are necessary and sufficient. Reexamination and careful notation of the faulty matches in alleged color blindness will almost certainly disclose simulation when it is attempted.

Asthenopia, vertigo and dizziness are a few of those complaints that cannot be denied if complained of by a patient. They bear the same relationship to a case as does a complaint of pain. These symptoms are present in both types of patients; they incapacitate both, and there are but few differences in them in the two conditions. Perhaps the hysterical subject is more likely to present these symptoms. The malingerer, however, does use them frequently. The neurasthenic patient is the one above all who wishes to exhibit them for consideration.

They represent a portion of his mental and physical fatiguability so characteristic of this neurosis. Adequate refraction, necessary vestibular testing and the various station and gait test might assist in unmasking the simulator, but one must be most careful that the interpretation of the reactions is correct.

The two conditions considered in this paper present so many features in common and are so different in others that all must be carefully studied for the sake of the patient, for purposes of fairness and in some instances as a rather interesting clash of wits.

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News and Notes

EDITED BY DR. JOHN HERBERT WAITE

UNIVERSITY NEWS

Prof. Emile de Grosz, on October 20, delivered a lecture at the Wilmer Ophthalmological Institute of the Johns Hopkins University on the "Operative Treatment of Glaucoma." He presented statistics from the enormous operative material of his clinic at Budapest as to the character of the glaucoma observed and the treatment employed. The outstanding feature in which his data differed from common experience in this country was in the high percentage of congestive types of glaucoma seen, amounting to 80 per cent of the total cases studied. In harmony with most of the continental ophthalmologists, he used the trephine operation in the majority of his cases until ten years ago. On account of late infections, trephining was then abandoned in favor of cyclodialysis and the Lagrange operation for most of his patients.

Miss Ida Mann, during the last week of October at the Wilmer Institute, gave a course of three lectures dealing with the "Development of the Human Eye." Her lectures consisted of a brief summary of the important data more extensively presented in her excellent book, and were profusely illustrated by drawings of the microscopic appearance of the human eye at various embryologic periods.

PERSONAL

William Mackenzie Award to Prof. J. van der Hoeve.—The William Mackenzie medal has recently been awarded to Prof. J. van der Hoeve of Leyden, Holland. On October 3, he delivered an address at the Glasgow Eye Infirmary on "Ophthalmology in Its Relation to the Other Branches of Medical Science."

GENERAL NEWS

The Need for Teachers in the Education of the Blind.—A circular letter from the Graduate School of Education of Harvard University calls attention to the following facts. In the United States there are 48 residential schools for blind youths, and 40 cities that conduct day classes for the blind in their public schools. The census returns list 863 teachers of the blind. Attention is called to the opportunities that exist in this special field for those who have a general educational background and who are willing to acquire an adequate specific training for the work. In addition to the educational interests in this work, there is also the medical interest and the social service interest. Blind children are generally more eager to learn and less distracted than school children in general. Classes for the blind are small, and instruction tends to be individual. Recompense is usually attractive and tenure secure. The departments of instruction are musical training, manual training and English branches through high school.

DEATHS

Dr. David Nicholas Dennis died of an acute attack of heart disease at his home in Erie, Pa., on November 22, at the age of 72. He was born in Grafton, Mass., in 1858. He obtained his education in the Grafton and Worcester schools, and finally entered Jefferson Medical College and was graduated in 1881. He started his practice in Philadelphia, but later went to Worcester. He finally settled in Erie, where he practiced for many years.

Dr. Dennis was a member of the American Medical Association, American Ophthalmological Society, Buffalo Ophthalmological Society and the Erie County Medical Society. He was also a Fellow of the American College of Surgeons.

William James Chapman, M.D., D.O.M.S., late major in the Canadian Army Medical Corps, died at the Western Hospital in Toronto. Born in 1864 at Belfast, Ireland, he graduated at the University of Toronto Medical School and practiced general medicine for over twenty-five years, at Kenora, Ontario. About eight years before his death he attended Moorfields, London, and ophthalmologic clinics at Vienna and other continental cities. On returning to Toronto, he became a member of the staff of the Toronto General Hospital and the Academy of Medicine. His cheerful good nature and willing cooperation endeared him to all his many friends and colleagues.

CORRECTION

In the report of the Section on Ophthalmology of the American Medical Association, in the October issue of the ARCHIVES, the following corrections should be made:

p. 585, line 16, Brich-Hudifield should read Birch-Hirschfeld.
p. 587, line 32, Gulen should read Grunert.
p. 587, line 37, eye should read myopia.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

THE SEPARATION OF THE LENS FROM THE ECTODERM IN MAN. F. FISCHER, Arch. f. Ophth. **124:** 507, 1930.

The process of the separation of the lens from the ectoderm was studied on eleven embryos of from 6.5 to 9 mm. in greatest length. By almost uniform proliferation of the cells at the margin of the lens pit a circular fold is formed which narrows the lens pore more and more. This proliferation continues until the margins of the circular fold touch each other. They then fuse and form two separate layers, the ectoderm and distal wall of the lens vesicle. In another mode of separation the margins of the circular fold do not reach each other, but the cells of the outer layers continue to grow until they finally fuse. The space in the distal wall of the lens is closed secondarily.

P. C. KRONFELD.

Conjunctiva

THE IMPORTANCE OF TERRAIN IN DISEASES OF THE EYE. L. WEEKERS, Arch. d'opht. **47:** 13 (Jan.) 1930.

In this paper Weekers discusses at length the influence of age and sex on the frequency of phlyctenular keratoconjunctivitis and the possible underlying causes. In a series of cases reported in 1911, 73 per cent of the patients were female, and in a larger series studied since 1919, 71 per cent were female. Except for the reports of Marfan and Grönholm in 1928, Weekers was unable to find reference to this predominance either in the special studies of phlyctenular disease or in the classic works. It is easy to verify clinically.

In the great majority of cases, phlyctenular disease is intimately connected with the process of immunization against tuberculosis which takes place in the early years of life in most persons. Weekers gives in detail the clinical evidence that supports this view as well as that which indicates that the tuberculous toxin is the causative factor.

He thinks that the phlyctenules observed clinically are due to the action on a hypersensitive conjunctiva of an infinitely small amount of tuberculin, of endogenous origin, diffused with the tears in the conjunctival sac spontaneously or at the time of some catarrhal condition, such as measles. The predominance of the disease in females can be explained on this basis in view of the work of Dujardin who has shown conclusively that the female reacts more strongly and more quickly than the male to the intradermal injection of the same antigen.

The relationship to age is then presented in a series of tables and graphs which lend further support to this view. In the first decade 56 per cent of the patients were female, the percentage immediately rising to 70 or more in subsequent years. Attention is called to the doctrine of Ranke and the conformity of the observations to it. The frequency of phlyctenular disease follows closely the variations in allergic hypersensitivity.

After dealing in considerable detail with phlyctenular disease the author points out similar relationships in the case of tubercular sclerokeratitis, tuberculous iritis, tuberculosis of the conjunctiva and the lacrimal gland and other ocular manifestations of tuberculosis. The article should be read in the original.

S. B. MARLOW.

Congenital Anomalies

A RARE CASE OF VERTICAL HETEROTOPY OF THE MACULAE. A. BIELSCHOWSKY, Klin. Monatsbl. f. Augenh. **84:** 755 (June) 1930.

Many cases have been described in which divergent or convergent strabismus seemed to be present or slight heterophoria became hidden, by the positive or negative condition of the angle gamma. Vertical aberrations have been reported only a few times. In the case described here a positive vertical divergence seemed to exist on fixation of the left eye, while only slight convergence appeared on fixation of the right eye. Examination with Bjerrum's board revealed the entire blind spot of the left eye to be located below the equator of the field of vision, and that of the right eye above the equator; the left macula was 13 degrees below, and the right 6 degrees above, the posterior pole. Both blind spots had the unusual distance of from 18 to 19 degrees from the center.

Old choroidal changes could be seen temporally, above in the right eye and below in the left. The author does not believe that the macula was misplaced by scar traction. The vertical distance of the images measured 13 degrees when the colored glass was placed in front of the right eye, but when it was placed before the left eye the distance was only 6 degrees; after a short while it rose to 13 degrees. This phenomenon corresponded to the downward motion of the right eye.

K. L. STOLL.

Cornea and Sclera

CHANGES IN THE POSTERIOR SEGMENT DUE TO HYPOPYON KERATITIS. DEL DUCA, Saggi di oftal. **5:** 113, 1930.

The observations of previous authors, who observed in sections of eyes with hypopyon keratitis swelling and infiltration at the posterior pole, especially in the nerve head, are reviewed.

The author reports ten such cases; the changes in the fundus were followed as well as the opacities in the media would permit, and the globes were sectioned after enucleation.

In all of the cases the vitreous showed cellular deposits among its fibers, which in some cases formed masses that extended toward the posterior pole. Their distribution showed no indication of a hyaloid canal as forming a channel for the spread of such cells.

The choroid was normal except near the nerve. The retina, however, showed small-celled infiltration in the more internal layers extending back to the papilla, which showed marked infiltration. This was especially marked about the retinal vessels and in the nerve fiber layer. The infiltration at the posterior pole consisted almost entirely of lymphocytes. No organisms were found in sections of the infiltrated tissue, or in the hypopyon, and animal inoculations from it were negative,

as were those from the ulcers. Apparently the products causing inflammation pass by the veins of the iris to the ciliary body, and thence to the posterior chamber and vitreous.

The infiltration of the nerve and the spaces about the central vessels, which was found in all cases, never reached the lamina cribrosa. The author believes that the damages are caused by toxic products transmitted by the vitreous.

S. R. GIFFORD.

THE BEHAVIOR OF DESCEMET'S MEMBRANE IN TRAUMAS AND OCULAR INFLAMMATION. I. NEUSCHÜLER, Saggi di oftal. **5:** 429, 1930.

The literature on changes in Descemet's membrane contributed before and after the introduction of the slit-lamp is reviewed. The author finds the present classification of such changes somewhat confused. He would distinguish between true folds and false folds of this membrane. True folds are parallel, and always at the same level. They may be due to both trauma and inflammation. Trauma that depresses the cornea produces the most typical true folds, long, with definite double contour, ending in points and seldom crossing. They are to be explained on a purely mechanical basis, and are usually transitory. Three such cases are described and illustrated. Trauma that opens the anterior chamber produces a type of true folds which are smaller and more numerous. These are produced by the release of traction on the membrane corresponding to the wound, and to resulting edema. Such folds are seen most commonly in the keratitis striata following an operation for cataract. Another group of true folds is found by inflammation, retraction of scars, changes in intra-ocular tension and changes in corneal curvature. The folds in glaucoma are due to edema of the cornea, and resemble inflammatory folds. Those due to hypotension, especially in phthisis bulbi, are very large and involve almost the whole thickness of the cornea.

The false folds due to inflammation are at various levels in the cornea, are not parallel but cross in all directions and are not so clear in outline as true folds. They are usually due to old exudate in the deeper layers between the corneal lamellae. Another type is due to exudate on the posterior surface of the cornea. These conditions as seen by the slit-lamp are illustrated by case reports and drawings.

S. R. GIFFORD.

Experimental Pathology

EXPERIMENTAL GLAUCOMA IN DOGS, WITH SPECIAL REFERENCE TO THE CHANGES OF THE OPTIC NERVE. F. ISHIKAWA, Arch. f. Ophth. **124:** 387, 1930.

Emulsions of paraffin oil were injected into the anterior chamber of dogs. About twenty-four hours later the tension went up, the whole picture being similar to that of acute inflammatory glaucoma in man. The eyes rapidly became ectatic, the posterior part almost to the same extent as the anterior part. In the course of a few weeks after the injection, the inflammatory symptoms and the increased tension gradually subsided. All the stages of this experimental glaucoma were studied anatomically (the earliest specimen 2 days, and the latest one 300

days, after the injection). The corneal epithelium was practically normal and the opacity which was observed clinically was caused by changes in the stroma. In several cases tears of Descemet's membrane were found. The root of the iris was adherent to the cornea in every case except one which showed an open angle but obliteration of the spaces of Fontana. In other experiments in which emulsions of human hair pigment were injected, the entire iris became adherent to the cornea. The retina was normal and the intralaminar part of the optic nerve showed cavernous atrophy in every specimen (even in the eye that was enucleated 2 days after the onset of glaucoma). The prelaminar part of the optic nerve showed the atrophy much later, a fact that speaks against Elschnig's theory of the histolytic power of the vitreous fluid in acute glaucoma.

In six of the author's experiments the development of a glaucomatous excavation was observed. It was caused by a recession of the lamina cribrosa and not by cavernous atrophy of the prelaminar and intralaminar part of the optic nerve. The excavation could not be considered a part of the ectasia of the eyeball. "The mechanism of the glaucomatous atrophy of the optic nerve is a stretching of the very delicate optic fibers, caused by a recession of the lamina cribrosa whose glial layer is intimately connected with the nerve fibers."

P. C. KRONFELD.

EXPERIMENTAL HYPERGLYCEMIA AND OCULAR CHANGES. N. COLLEVATI, Saggi di oftal. **5:** 85, 1930.

The author produced hyperglycemia in dogs by various methods, and studied the ocular changes produced. Injections of epinephrine and morphine produced a glycemia of from 2.34 to 3.34 mg. per thousand cubic centimeters before death, the aqueous in the latter case containing 1.8 mg. per thousand cubic centimeters of dextrose. Injection of pilocarpine produced less marked hyperglycemia. Dogs with partial pancreatectomy did not survive longer than from three to five days, and their glycemia was not greatly increased.

All of the animals showed changes in the ciliary body and lens. The ciliary epithelium showed marked degeneration; the pigment epithelium was swollen and granular, and the vessels were engorged.

The sugar content of the aqueous was increased in dogs with hyperglycemia, without ever reaching the sugar concentration of the blood. The lenses showed marked vacuolization of the fibers without going on to complete opacification. No lesions of the posterior segment were observed. This suggests differences between experimental hyperglycemia and diabetes, in which other factors probably play a part.

S. R. GIFFORD.

Glaucoma

THE SURGICAL TREATMENT FOR CHRONIC GLAUCOMA. C. DUVERGER and E. VELTER, Arch. d'opht. **47:** 5 (Jan.) 1930.

The authors consider only those cases in which no known cause can be demonstrated to explain the hypertension as primary chronic glaucoma. They emphasize the necessity of slit-lamp examination in

the study of these cases. Therapeutic progress will consist in the progressive dismemberment of so-called primary hypertension through the discovery of many diverse etiologic factors that can produce a pathologic process with elevation of tension. Until that time is reached, operation will be necessary. The authors do not hesitate to resort to the Lagrange operation when the glaucomatous process progresses in spite of miotics and well conducted medical treatment.

They summarize the important points in their method of performing the Lagrange operation, which they believe to be the procedure of choice: (1) the cutting of the scleral and conjunctival flaps with a Graefe knife by puncture and counterpuncture 1 mm. behind the limbus, the length of the incision being from 5 to 6 mm. at the maximum; (2) the careful dissection of the conjunctiva up to the limbus with a Graefe knife; (3) the resection of the scleral lip with a Graefe knife, the edge directed backward and downward, and (4) peripheral iridectomy.

The authors are of the opinion that a properly performed Lagrange operation is at the present time the best surgical means of treating patients for chronic simple glaucoma. The remote results are better than those of iridectomy in acute glaucoma.

S. B. MARLOW.

THE RÔLE OF THE SYMPATHETIC IN GLAUCOMA. J. BISTIS, Arch. d'opht. **47:** 96 (Feb.) 1930.

After a review of the theories proposed to explain glaucoma and a discussion of the objections to them, the author presents the results of some experiments on rabbits. These experiments lead him to believe that excitation of the sympathetic can produce a transitory elevation of the intra-ocular tension and intervene as the first step in the development of glaucoma. In order that this condition be established, however, a suitable terrain is necessary, prepared by local and general conditions, such as rigidity of the sclera, errors of refraction, arteriosclerosis and endocrine disorders.

S. B. MARLOW.

Injuries

AN UNUSUAL COMPLICATION OF TRAUMATIC CATARACT. T. W. LETCHWORTH, Brit. J. Ophth. **14:** 450 (Sept.) 1930.

The author reports a case in which a piece of steel was present in the lens, unsuspected by the patient. Later the matter in the lens, which had become opaque as a result of the injury, suddenly exuded into the vitreous. Lying embedded in the substance of the lens could be seen a glistening, metallic foreign body. A preliminary iridectomy was performed.

A month later sudden pain was experienced, with a slight rise in tension. The opaque lens could be seen in the vitreous chamber, below and behind the pupil.

The lens, piecemeal, together with the foreign body was removed by a vectis. The absence of evidence of the steel having entered through the cornea lends support to the view that the foreign body entered the lens through the posterior capsule, and through this opening the matter in the lens apparently escaped into the vitreous.

W. ZENTMAYER.

Lens

CATARACT IN DIABETES AND TREATMENT WITH INSULIN. PUSCARIU, Soc. Român. de oftal., Bucuresti **11:** 34 (Nov.) 1929.

A girl, aged 16, with diabetes of three and one-half years' standing and cataract in both eyes of six months' standing, was very weak, having glycemia of 4.5 in 1,000 and glycosuria of from 150 to 180 Gm. in twenty-four hours. After injections of insulin (20 units a day for five days successively) the sugar disappeared and the patient could be operated on with good result.

N. BLATT.

Neurology

SYMPTOMATOLOGY OF TUMORS OF THE OCCIPITAL LOBE. E. DE ANGELIS, Riv. oto-neuro-oftal. **7:** 241 (May-June) 1930.

In tumors of the occipital lobe, the only localizing sign is given by a hemianopia, which, however, is not at all specific for tumors of this lobe, for it is also observed with tumors at any point in the optic pathways between the occipital cortex and the chiasm. Only on a basis of concomitant symptoms is it possible to formulate a precise diagnosis of the locus in cases of tumor with hemianopia.

More often a tumor of the occipital lobe simulates, according to the direction of its growth, either a cerebellar tumor (if it extends toward the cerebellar fossa), a tumor of the temporal or parietal lobe (when it grows forward) or a tumor of the rolandic motor radiations (when growing toward these). The presence of hemianopia will save one from the error of making the diagnosis of tumor of the cerebellum; disturbances of sensibility, especially of deep sensation, will prevent confusion with neoplasms of the parietal lobe; disturbances of smell or taste, homolateral signs of paresis of the third nerve, associated with controlateral hemipareses and hemihypo-esthesias (Mingazzini), will identify tumors of the parietal lobe. If, however, the lesion is on the left side, these errors are more easily eliminated by the appearance of aphasic disturbances.

It is a rare occurrence for a tumor to reside entirely in the occipital lobe. More often there is an associated new growth involving the occipital and temporal or the occipital and parietal lobes. In such cases there can be no reliable clinical deduction as to which one of these lobes the tumor began in. Even the chronology of symptoms is not always helpful, since hemianopia may occur as a first symptom also in tumors occupying the medullary substance of these lobes.

VICTOR R. SYRACUSE.

Ocular Muscles

SOME ORIGINAL EXPERIMENTS WITH THE O'CONNOR MUSCLE-SHORTENING OPERATION. J. L. MCCOOL, Am. J. Ophth. **13:** 491 (June) 1930.

This is an experimental study of the amount of shortening effected by the original O'Connor operation. The author found that each strand of fine dermal suture will effect a shortening of 0.55 mm. when lateral strips of tendon measuring from 2 to 2.5 mm. in width are used. With a medium dermal suture, the shortening per strand is 1 mm. When the

lateral strips of tendon are 3 mm. wide, the shortening is 0.66 and 1 mm. plus, respectively. O'Connor's original technic was used, with the exception that a dermal suture was substituted for catgut.

W. S. REESE.

NERVE PARALYSIS FOLLOWING LUMBAR ANESTHESIA. V. POPOVICI, Vita med. **5:** 304, 1929.

Palsy of the fourth nerve after lumbar anesthesia is exceedingly rare. Thus far only two cases are said to have been published, the author's case being supposedly the third. The unilateral palsy of the fourth nerve appeared ten days after lumbar anesthesia had been applied. Syphilis could be ruled out owing to the negative Wassermann reaction and to the negative reaction after administration of neoarsphenamine. Prognostically, the author emphasizes the fact that diplopia persisted after nine months. When he had the opportunity to see the patient after five years, the latter presented no subjective or objective symptoms. The direct cause of the palsy was ascribed to the pressure the hematoma exerted on the nuclear area or filamentous area of the corresponding nerve. The indirect cause was due to a meningeal reaction elicited by the solution used (benzoyldimethylaminoethylpropanol hydrochloride, cocaine). The predilection of these palsies for certain nerves seems to be connected with the anatomic site of the vessels of these regions.

N. BLATT.

Operations

OPTIC NERVE SURGERY. D. M. VELEZ, Am. J. Ophth. **13:** 408 (May) 1930.

The author discusses optic neuritis and strangulation of the optic nerve, and proposes an operation for exposing the optic canal and taking out the fragment forming the upper part of the canal. The various steps of the operation, as worked out on cadavers, are described.

W. S. REESE.

OPERATIVE REMOVAL OF HERNIAS OF FAT ON THE LOWER LIDS. A. ELSCHNIG, Klin. Monatsbl. f. Augenh. **84:** 763 (June) 1930.

Subsequent to his description of various types of disfiguring bags of the lower lids, the author reports his method of removing them in a woman, aged 20, a dancer. The epidermis of the lids was normal; the size of the bag could be greatly increased by pressure on the globe. The eyes were operated on, a week apart, under local anesthesia. A curved incision was made along the fold of the lower lids. i. e., along the upper margin of the bag, and the epidermis was separated from the orbicular muscle up to the lower margin of the fatty pad. After the fascia of the orbicular muscle had been opened, parallel to the orbital margin, on slight pressure on the globe the fat presented for a length of 2 cm. It was adherent neither to the tarso-orbital fascia, nor to the orbital fat. Great stress is laid on the incision at the upper margin of the fatty pad, on the incision into the fascia at its lower margin, as described, and on the care in laying the sutures. After applying two deep sutures, the incision of the skin is closed by closely arranged

sutures of the finest material. The knots are tied loosely and removed on the third or fourth day, thus leaving no marks. Ligation of the blood vessels should be avoided, and all bleeding stopped carefully. Secondary hemorrhages should be prevented by a properly applied bandage. In the case reported, a fine linear scar was all that could be seen when the bandage was removed. K. L. STOLL.

Pharmacology

ON THE ACTION OF PUPILLOMOTOR DRUGS IN LOCALIZED APPLICATION. F. GNAD, Klin. Monatsbl. f. Augenh. **84:** 510 (April) 1930.

Experiments were made by the author at Elschnig's clinic at Prague with atropine, homatropine, cocaine, glaucosan (an epinephrine substitute), eserine and pilocarpine. A small mop of cotton, moistened with solutions of these drugs, was applied at certain parts of the limbus after the instillation of a local anesthetic, *di*-allyloxyethyl-*di*-phenyl amidine hydrochloride; cocaine with epinephrine was injected under the conjunctiva. The experiences gathered permit an explanation for the greater frequency of prolapses of the iris after subconjunctival injection of cocaine with epinephrine, as compared with anesthesia by retrobulbar injection. Subconjunctival injections, therefore, should be avoided at the site of the incision whenever the iris is to remain intact. Every reacting pupil can be influenced by this local application, and it will return to its original round form. The form and extension of the dilatation depend on the duration and technic of the application; they are influenced by the elasticity of the iris and the age of the patient. Glaucosan (an epinephrine substitute) is considered useful in the selection of the site for an optic iridectomy because it produces a markedly localized slit-shaped dilatation without involving the adjacent areas of the iris. K. L. STOLL.

The Pupil

A CASE OF CORECTOPIA. A. A. KNAPP, Am. J. Ophth. **13:** 141 (Feb.) 1930.

A Porto Rican boy, aged 5, whose personal and family history was irrelevant, showed eccentric pupils temporally and somewhat superiorly. The nasal side of each iris was arranged in radiating folds and appeared to be stretched toward the pupil. To the temporal side of each pupil was a band of iris tissue. Over the temporal portion of the pupillary area of each cornea was a vertical linear opacity in which the iris was adherent. Tension in the right eye was 85; in the left eye, 70 (McLean). W. S. REESE.

Physiology

COMPARATIVE STUDIES ON THE PERIPHERAL AND CENTRAL RETINA. RAGNOR GRANIT, Am. J. Physiol. **94:** 41 (July) 1930.

The author has studied the interaction between distant areas in the human eye. He measured the fusion frequencies with the Flicker method when the periphery of the retina was stimulated at one or more points simultaneously. By measuring the fusion frequency of four circular test patches separated from each other by an unstimulated area

it was possible to determine whether there is an interaction between distant areas of the human eye similar to that found by Adrian and Mathews who recorded the impulses from the optic nerve of the conger eel. With central fixation there was practically no interaction found even with high intensity of illumination. With peripheral fixation, on the other hand, there was an increase in the fusion frequency demonstrating interaction between the various stimulated areas even when the intensity of the stimuli was diminished 100 times.

The fact that contours are normally blurred in peripheral vision has been known to be due in part to defects of the refractive system. The present experiments, however, show that the blurred contours in the periphery are due in part to a physiologic spread of excitation. The peripheral retina of the human eye closely resembles the primitive retina of the conger eel in structure, whereas the human fovea is differently composed. The difference between central and peripheral vision with regard to interaction corresponds with the increasing structural differences in the two parts of the retina.

F. H. ADLER.

THE MECHANICAL THRESHOLD OF THE CORNEA-REFLEX OF THE USUAL LABORATORY ANIMALS. H. STRUGHOLD, Am. J. Physiol. **94:** 235 (July) 1930.

The author has studied the threshold value of the corneal reflex in rabbits, guinea-pigs, dogs, cats, pigeons, rats and monkeys, using the test hairs of von Frey. Dogs, cats and monkeys were found to be fairly unsatisfactory subjects because of their restlessness. The thresholds found were as follows:

Species	Threshold, Gm. per sq. mm.
Rabbits	8–10
Cats	5–6
Dogs	4–5
Pigeons	4
Rats	1–1.5
Monkeys	0.5–1
Man	0.2–0.3

Like man, the animals also showed the center of the cornea to be much more sensitive than the periphery. Strughold expresses the belief that the high sensibility of the center of the cornea depends on the richness in nerve endings of this part of the corneal epithelium compared with the marginal zones. It follows that in comparing the threshold values it is necessary always to stimulate the same relative part of the cornea. The values given in the table are the threshold values for the center of the cornea.

F. H. ADLER.

THE RESULTS OF EXTIRPATION OF THE SUPERIOR CERVICAL GANGLION ON THE UNSTRIPED ORBITAL MUSCLE OF THE RAT. ROBERT DINOLT, Arch. f. d. ges. Physiol. **224:** 619, 1930.

The superior cervical ganglion was removed from sixteen rats and the preganglionic fibers of the sympathetic cut through in five others. The paradoxic symptoms occurred only after extirpation of the gang-

lion. They depend on a peripheral hypersensitivity. After the operation there was enophthalmos and narrowing of the palpebral fissure on the side operated on. From twelve to eighteen hours later, during narcosis, the lids became wider; twenty-four hours later there was exophthalmos. These signs occurred only during narcosis since the striped muscles are paralyzed in this condition and the peripheral hypersensitivity is made manifest.

On the other hand, the pupil dilated after twenty-eight hours with and without narcosis since it was more sensitive to slight changes in innervation. Ether, chloroform and magnesium sulphate were used as narcotics. These symptoms were further investigated after the use of atropine, physostigmine, epinephrine and ergotamine. Atropine dilated the pupil on the side operated on more rapidly than that on the normal side when the animal was narcotized. Physostigmine, on the other hand, showed no change from its action on the normal eye. Epinephrine, which showed no effect on the normal rat's eye, either by instillations or subcutaneous injections, produced a definite mydriasis forty-eight hours after the operation. Ergotamine produced a more marked constriction of the pupil on the paradoxically dilated side.

F. H. Adler.

Refraction and Accommodation

The Mechanism of Accommodation and Diabetic Myopia. A. Hudelo, Arch. d'opht. **47:** 70 (Feb.) 1930.

The observation of a case of acute myopia during the course of diabetes led the author to speculate on the mechanism of accommodation in general.

In studying the question in relation to diabetes he says that the conclusion will be quickly reached that myopia and hyperopia occur with about equal frequency in this disease. He therefore suggests that a single hypothesis should be able to explain both phenomena. The problem of changes in refraction in diabetes is usually considered either as of a physicochemical or as of a paralytic nature. Under the general heading of the physicochemical hypothesis he discusses the relationships of the dextrose content of the intra-ocular fluids and the hydration or dehydration of the globe as the glycemia rises and falls; he goes to some length in his argument in disagreement with the proposals of Duke-Elder. The theories of lengthening or shortening of the globe under the influence of choroidal edema he does not consider nearly so worthy of notice as those relating to the lens itself. The objections to these are cited.

The dynamic theories *a priori* are equally unsatisfactory because they would not explain myopia and can be applied only to hyperopia. The explanation of myopia by a dynamic theory would necessitate the inducing of ciliary spasm. Diabetes results only in paralytic muscular changes. Hudelo rejects the conception of myopia in diabetes as the result of spasm for the following reasons: 1. As diabetes never produces muscular spasm or contraction, the ciliary spasm would have to be an exception. 2. If the myopia was due to spasm, atropine should relieve it, whereas the majority of patients are uninfluenced by it. 3. If there were toxic spasm, there would always be frontal headache. 4. True

spasm of accommodation nearly always produces a high degree of myopia whereas the myopia found in diabetes rarely exceeds 3 diopters.

After reviewing the theories of Helmholtz and Tscherning he describes a dualistic hypothesis. The anatomic basis for this idea is the presence of two opposing muscles which make up the ciliary body: the sphincter composed of circular fibers and Brücke's muscle consisting of radiation fibers.

Brücke's muscle is well developed in persons in whom accommodation is relaxed, in myopic persons and in those whose accommodation is paralyzed, e.g., by atropine (Heine). The sphincter muscle is developed in those who exert their accommodation, in hyperopic persons and in those whose eyes are stimulated by physostigmine (Heine). The physiologic basis for this theory lies in the established fact of a dual nerve supply, the sphincter being innervated by the oculomotor nerve and the radiating fibers by the sympathetic nerve. This corresponds to the physiology of muscles elsewhere in the body. Helmholtz' theory is made use of to explain the action of the sphincter when it is stimulated. The action of Brücke's muscle is as a tensor of the choroid by means of which a pull is put on the zonule and pressure is exerted on the vitreous so that the lens is flattened. In near vision the sphincter contracts to relax the zonule, allowing the lens to become more spherical while the radial muscle relaxes. In distant vision the sphincter relaxes while the radial muscle draws on the zonule and increases the vitreous pressure to flatten the lens. This explains the normal relation of the volume of the two muscles, 9:1. The sphincter, because of its more advantageous position is smaller; the radial muscle must be larger to have an equivalent effect because its action is an indirect one.

The author then makes use of this theory to explain not only the changes in refraction that may take place in diabetes but also those which occur in diphtheria, poisoning from arsphenamine, morphia and carbon disulphide, traumatic myopia, acute myopia of unknown cause and the myopia that may occur in convalescent persons.

He summarizes his paper in tabular form from which the following is translated:

Paralysis of the sphincter plus integrity of the tensor choroidia equals hyperopia (always slight).

Paralysis of the tensor plus integrity of the sphincter equals moderate myopia.

Paralysis of both equals loss of accommodation with conservation of the refraction in its original state.

Parcellary paralysis equals astigmatism.

Hudelo also reports a case of acute myopia in diabetes. His paper deserves more attention than an abstract can give to it.

S. B. MARLOW.

Retina and Optic Nerve

ETIOLOGY OF PAPILLEDEMA (CHOKED DISK). CARLOS CHARLIN, Arch. de oftal. de Buenos Aires **5:** 122 (March-April) 1930.

The author protests against the custom of referring cases of papilledema to the ophthalmologist without clinical data as to the general condition of the patient. Internists simply demand the results of

the examination of the eye to add to their protocols. He considers this line of conduct as disastrous, as choked disk leads to blindness, as it is primarily a disease of the eye, the treatment for which should be directed by the opthalmologist, who should be supplied with all the available data as to the condition of the body outside the eye.

The article is based on the observations in fifty-two cases, which have been studied with neurologists, internists, pediatricians, and others. The fifty-two cases can be classified etiologically as follows:

Nineteen cases due to tumors (of these one was a hematic cyst and two were tuberculomas); nineteen cases due to syphilis (of these seven were gummas); seven due to uremia; two due to tuberculous meningitis, and five produced by other causes.

The author points out the following characteristics in cases of tumor: focal symptoms in the shape of hemiplegia and basal, cortical, cerebellar and psychic symptoms, which were noted in sixteen of the nineteen cases; the exceptions were lack of focal signs in a case of tumors (two) in the frontal lobe and in both cerebellar lobes, in cases of tumor of the central lobe of the cerebellum (vermis) and in a hematic cyst of the cortex; complete helplessness on the part of the patients, and complete integrity of the cardiovascular system. The examination of the cerebrospinal fluid in these cases always showed a pronounced hypertension, with a high albuminosis and a low leukocytosis (Sicard's syndrome), and a negative Wassermann reaction (it is exceptional to find a nonsyphilitic tumor giving a positive Wassermann reaction).

The syphilitic cases comprise two groups: cases with focal signs (gummas) and cases without focal signs (meningitis). The first group shows the same symptoms as the tumor group, with marked changes in the nervous system of a focal nature, with the same helplessness and integrity of the cardiovascular apparatus. The spinal fluid shows a high leukocytosis and a positive Wassermann reaction. In a few cases, in which some doubt may exist, a trial treatment may be instituted, the syphilitic cases yielding promptly to specific treatment. The second group, without focal symptoms, comprises two classes: cases with simple cerebrospinal hypertension with a negative Wassermann reaction and cases with a high leukocytosis, a high albuminosis and a positive Wassermann reaction. This group is also characterized by its response to appropriate treatment. These cases, if not treated, terminate in blindness.

The uremic cases present headaches, vomiting and choked disk. Lumbar puncture shows a high increase of tension. There may be a meningeal reaction, with some albuminosis and a slight leukocytosis, and at times Sicard's syndrome. The principal characteristics are high arterial hypertension, renal insufficiency and a poor general condition. There is always a pronounced pathologic condition of the cardiovascular and renal system. Treatment should be controlled by diet, blood-letting and repeated lumbar punctures.

In the group of tuberculous cases, there also are cases with focal symptoms and cases with no focal localization. There is generally a high temperature, a poor general condition, a great meningeal reaction, albuminosis, a high leukocytosis and a reduction of chlorides. At times Koch's bacillus can be found in the cerebrospinal fluid.

Of the other cases, one was due to an infectious meningitis of tonsillar origin; in two no etiologic factor could be demonstrated, and one was due to eclampsia.

The author finally condenses these observations in a schematic chart.

C. E. FINLAY.

Tumors

MELANOSARCOMA AT THE LIMBUS. J. W. McKEE, Am. J. Ophth. **13:** 407 (May) 1930.

A woman, aged 31, presented a deeply pigmented, almost black, extremely vascular growth at the temporal limbus. Three months after two applications of the thermophore, all trace of the tumor had disappeared, and vision was 6/6.

W. R. REESE.

HAEMANGIOMA OF THE ORBIT. T. H. CRESSWELL and W. A. BRIGGS, Brit. J. Ophth. **14:** 357 (July) 1930.

A woman, aged 26, gave a history of a defect of vision with an increasing prominence of the left eye. The movements of the globe were full. The globe could not be pushed back. There was moderate papilledema and hyperopia of 5.5 diopters.

A curved incision was made through the conjunctiva close to the fornix to the outer side and the external rectus was divided. On the introduction of a finger behind the globe the tumor was suddenly delivered, jumping a foot from the wound. The pathologic report made by Winter was that the specimen was heart-shaped, liver-colored and surrounded by a thin glistening capsule. The tumor measured 2.5 by 2.5 by 1.5 cm. The contents seemed to consist of innumerable small spaces, separated by well formed tissue and containing blood. Microscopically the section showed irregular communicating spaces the walls of which were lined with endothelial cells and which were filled with blood vessels.

W. ZENTMAYER.

TWO CASES OF INTRA-OCULAR SARCOMA. H. GRIMSDALE, Brit. J. Ophth. **14:** 410 (Aug.) 1930.

The first case occurred in a single woman, aged 54. Eight years previously, the left breast had been removed because of carcinoma. The left eye showed a solid detachment of the retina. The mass was more or less hemispherical, projected forward considerably and was not much pigmented.

In view of the history, it was thought probable that the growth was a carcinoma, but histologically it proved to be a sarcoma. The author feels that the proper diagnosis would have been made had he not been obsessed by the history.

The second case occurred in a married woman, aged 70, whose right eye was the seat of iritis with hyphemia. On subsidence of the inflammation, there was seen a dark brown mass pushing through the base of the iris and flattened against the cornea.

W. ZENTMAYER.

PROGNOSIS IN CASES OF UVEAL SARCOMAS. E. VON HIPPEL, Arch. f. Ophth. **124:** 206, 1930.

Few conclusions can be drawn from statistics on cases of sarcoma of the uvea. Of von Hippel's patients, 17 per cent died from five to ten years following enucleation, and 10 per cent died after ten years. From these figures it is seen that there is no time limit after which a case can be considered as cured. In von Hippel's material metastatic tumors occurred in 4 per cent of the cases. Even this figure is too low in von Hippel's opinion. He also believes in a relative immunity against the formation of metastatic tumors. The spread of sarcoma cells all over the body is undoubtedly more frequent than the occurrence of metastatic tumors. Von Hippel found a large number of cases in which tumor cells could be seen in the emissaria of the eyeball. Twelve of these patients lived for more than five years. Tumor cells in the emissaria are not always a bad prognostic sign.

Von Hippel performs enucelation in all his cases of sarcoma as soon as the diagnosis is made, provided that the other eye has useful vision. If the eye with the sarcoma is the only seeing eye, von Hippel waits as long as the vision of this eye remains useful. P. C. KRONFELD.

Uvea

TWO CASES OF EPITHELIAL IMPLANTATION CYST OF THE IRIS. R. F. MOORE, Brit. J. Ophth. 14: 496 (Oct.) 1930.

The first case was incompletely reported in 1924 by R. C. Davenport (*St. Barth. Hosp. Rep.* **37:** 141, 1924). The right eye of a house surgeon was cut with spectacle glass. The wound was linear and extended obliquely across and through the middle of the cornea. There was no prolapse of the iris. The lens was uninjured. The next day, on the front of the iris at 4 o'clock, at the site where the cyst afterward formed, was a small grayish-white, rather woolly looking mass. Later a few fine keratitic deposits were seen. Though vision returned to 6/6, the eye was soft and irritable so that an intra-ocular foreign body was suspected but not shown by the roentgen rays.

Ten months later, at the inner end of the scar was a small, spherical, pearly cyst, and lying across it with its root uppermost was an eyelash. Four months later the lash was removed, along with the cyst, by iridectomy. The lash showed no evidence of growth. Five months later a second, precisely similar cyst developed in the angle of anterior chamber, situated exactly in the coloboma as if in connection with the cut root of the iris or the ciliary body. It was dissected out and removed. Eight years after the accident the visual acuity was better than 6/6, and the eye caused no trouble.

The second case was that of a woman, aged 48. A piece of coal penetrated the cornea at the limbus and lodged on the iris, from which it was removed through a keratome incision. Later a small woolly spot appeared on the iris. About five months later a small pearl-like cyst with an eyelash across it was present. It was caused to prolapse outside the globe through a corneal incision and removed. The eye has remained quiet since.

The pathologic diagnosis was implantation epithelilal cyst of the iris. Points of interest were: in both cases the lash was unattached to the cyst; by dissecting up a conjunctival flap which was attached to the cornea, the wound was made to gape by pulling on the flap and using it as a handle, so that the cyst protruded prominently from the wound; the presence of the woolly looking fragment on the iris in both cases, which afterward seemed to disappear as if in some way it was a precursor of the development of the cyst.

W. ZENTMAYER.

PERFORATING SOLITARY TUBERCLE OF THE CHOROID. NICOLAU, Soc. Român. de oftal., Bucuresti **11:** 33 (Nov.) 1929.

A choroidal tumor in the equatorial region broke through the sclera at the end of five weeks. There were hypotonia, plastic iritis and almost total detachment of the retina. Vision was reduced to the counting of fingers at 20 meters. Tuberculosis was mentioned frequently in the family history. Histologic examination showed typical tuberculous follicles; there was no evidence of tubercle bacilli. In spite of this severe condition, treatment by ultraviolet rays produced a striking improvement due to the absorption of the tumor, the regular cicatrization arising between the sclera and the choroid and the (spontaneous) reattachment of the retina.

N. BLATT.

TWO CASES OF IRITIS ASSOCIATED WITH MUMPS. V. POPOVICI, Viata med. **5:** 56, 1929.

Seven cases of iritis—iridocyclitis following epidemic parotitis—are said to have been published in the literature. The author describes two such observations, one in a girl aged 8 years, and one in a woman aged 20. The iritis began from four to five days after the mumps had begun. The nature of the disease was benign. Therapy consisted of instillations of atropine and intravenous injections of neoarsphenamine.

N. BLATT.

Sympathetic Ophthalmia

THE FIRST STAGES OF SYMPATHETIC OPHTHALMIA DETECTED BY BIOMICROSCOPY: PRACTICAL RESULTS OF EARLY DIAGNOSIS. JEANDELIZE and BRETAGNE, Arch. d'opht. **47:** 65 (Feb.) 1930.

Although Vogt has already described some cases of fugacious sympathetic ophthalmia, the authors call attention to similar instances because of the great necessity of making an early diagnosis, which can be done only by biomicroscopy. Their observations support the conclusions of Vogt: (1) that there is a fugacious type of sympathetic disease which cannot be detected by ordinary clinical methods; (2) that so-called "sympathetic irritation" is in reality a more advanced stage than the mild fugacious type, and (3) that these mild reactions must be recognized, watched and treated with great care so as to prevent if possible the usual classic form of the disease.

S. B. MARLOW.

Therapeutics

THE USE OF THE DIATHERMY CURRENT IN EYE DISEASES. P. FIGDOR, Brit. J. Ophth. **14:** 405 (Aug.) 1930.

The action of diathermy of the eye can be divided into two parts: heat, for relief from pain (and perhaps also a factor in the actual treatment for the disease process, though this is a difficult matter to prove or disprove), and hyperemia, as the primary therapeutic factor of the process.

The method of applying the bath is described. It is advised that the electrode be applied dry. The eye may be opened or shut. The first treatment should last four minutes as a maximum; subsequently, the maximum may be six or seven minutes. Usually, two treatments are given weekly. The patient must not be left for a moment, and the hand should be kept close to the switch. The impression that the author has received is that pain in the eye from whatever cause in any chronic disease of the eyes is always relieved by diathermy. Patients with chronic senile conjunctivitis invariably do well. Patients with cyclitis, iridocyclitis or episcleritis are usually benefited. In glaucoma the tension is appreciably lessened after each treatment, at least temporarily, and in some cases the condition appears to be arrested. Most cases of cataract did not respond to treatment, but in one case resolution seemed to have become complete with restoration of almost full vision. The author deprecates any extravagant claims for diathermy.

W. ZENTMAYER.

ALCOHOL INJECTION AS TREATMENT FOR BLEPHAROSPASM AND SPASTIC ENTROPION. KARL SAFAR, Ztschr. f. Augenh. **71:** 135 (May) 1930.

An injection of from 2 to 3 cm. of 80 per cent alcohol into the deeper tissues of the temporal area of the orbit gives considerable relief from the discomforts produced by blepharospasm. Care must be taken to avoid injection of the alcohol into the subcutaneous tissue, for a burning sensation is caused by this mishap. An injection of from 2 to 3 cm. of procaine hydrochloride often alleviates pain produced by the subcutaneously injected alcohol. As a rule, the tissue of the lid becomes edematous and remains in this condition for two or three days. This reaction is of no consequence. In treating spastic entropion, a small amount of alcohol is used. It is best to inject it at the lower area of the orbital margin. The treatment is simple and does not necessitate hospitalization.

A. M. YUDKIN.

RESULTS OF ROENTGENOTHERAPY IN CERTAIN OCULAR DISEASES. DI MARZIO AND SALVATORI, Saggi di oftal. **5:** 5, 1930.

The authors have used the roentgen rays therapeutically in a large series of cases and report their results. They believe that with modern improvements in technic and standardization of dosage such therapy is without the dangers to the eye previously feared, and that it is of distinct advantage in certain conditions. In general, they employ feeble dosage, from 10 to 30 per cent skin erythema dose, in inflammatory

conditions such as corneal ulcer and interstitial keratitis, moderate dosage in trachoma and high dosage, 100 per cent skin erythema dose or more, in neoplasms. Hard rays, filtered through a 0.5 mm. zinc and a 4 mm. aluminum filter were used, and the doses were carefully calculated. Eighty-six patients with corneal ulcer were treated, with 73.5 per cent cures and 17.5 per cent improvement, the fascicular type of ulcer which resists other measures, responding especially well. From 10 to 20 per cent skin erythema dose was given every five to seven days for from two to five treatments. Forty-two patients with interstitial keratitis were similarly treated, with cure in thirty-eight per cent and marked improvement in 54 per cent of the cases due to syphilis. Healing was more rapid in these patients than in those not given roentgen rays. Antisyphilitic treatment was not given until after the roentgen ray treatments. Forty-six patients with uveitis, twenty-eight of whom were considered tuberculous, gave similar reactions, some good results being obtained in old cases which had resisted all other treatments. In sixty cases of trachoma from one-half to one-third skin erythema dose was given at intervals of from ten to sixty days, often combined with iontophoresis; in the older cases, scarification was performed. There were only 18 per cent of clinical cures, while the symptoms in many cases were improved. Some additional cases of trachomatous pannus showed considerable improvement. Of the thirteen patients with neoplasms who were treated, the results were most notable in two of five with retinocytoma (retinoblastoma); both of these showed destruction of the tumor in the second eye with preservation of useful vision during an observation of five months in one case and four years in the other. From 160 to 200 per cent skin erythema dose was given, divided over several sittings. No results were seen in four cases of vernal catarrh.

It is remarkable that even with the fairly large doses given for neoplasms, the authors observed no complications, such as cataract, due to the treatment.

S. R. GIFFORD.

THE MODERN THERAPY OF HYPOPYON KERATITIS. I. NEUSCHÜLER, Saggi di oftal. **5:** 316, 1930.

The author describes three principal methods used especially in the clinic at Rome. Roentgenotherapy, as described previously by di Marzio, has given excellent results, and in small doses, the author believes, aids in clearing the resulting scars. Ultraviolet light, administered by the Koeppe irradiation lamp, has also given good results, especially in early superficial ulcers, but has no effect on the scars. The third method, first used by di Marzio, is that of auto-aqueotherapy, by which is meant the subconjunctival injection of aqueous removed with a syringe from the affected eye. This is done with the idea that some protective substance is present in the aqueous which exerts a bactericidal effect when so used, and which in some way stimulates healing, though the author admits that the removal of the aqueous itself is of benefit. The procedure may be repeated every few days if necessary. Paracentesis alone is often employed, as is improves the nutrition of the cornea on account of the lowered tension, and as it brings a new supply of antibodies into the aqueous. When this method, in addition to the other methods, fails to prevent the progress of the ulcer, fistulization of the

anterior chamber is performed by opening the paracentesis wound repeatedly with a spatula. Foreign protein was used in some cases, a casein-calcium combination being preferred to milk.

Twenty-seven cases are reported, in which various combinations of the foregoing methods were employed. Only two eyes were enucleated on account of failure to check the infection, a third being removed two months after healing on account of secondary glaucoma. Some of the milder cases healed after two or three days following roentgenotherapy, ultraviolet irradiation or auto-aqueotherapy. In two cases in which panophthalmitis seemed to be developing, the condition healed under treatment. The author believes that in most cases the course of healing was materially shorter and the scars less dense than would have been expected with the usual methods of treatment.

S. R. GIFFORD.

EXPERIMENTS IN TUBERCULIN THERAPY. CARLOS PASTOR BAMBACH, Arch. de oftal. hispano-am. **30:** 354 (June) 1930.

These experiments were carried out in Charlin's Clinic in Santiago de Chile, according to Viton's method of tuberculin diagnosis and tuberculin therapy; he employs for this purpose infinitesimally small doses, commencing at times with dilutions of 1 : 100,000,000,000,000,000,000,000,000,000,000,000. Although he has employed this treatment in a comparatively large number of cases, the author reports only fourteen, which he was able to follow up for some time. Seven were cases of inflammations of the iris or ciliary body, three of scleral lesions (two with corneal complications), one of phlyctenular keratitis, one of phlyctenular conjunctivitis and two of typical tuberculous lesions of the conjunctiva. The cases are reported in detail, and the results were satisfactory in twelve cases, six patients being cured and six being notably improved. The treatment had no effect on the typical lesions of the conjunctiva, in accordance with Pinticart's experience.

The author's conclusions are: Tuberculous diseases of the eye are relatively frequent and generally present no characteristic features. The diagnosis is difficult and is obtainable only by the use of tuberculin. Mantoux's cutaneous reaction is of value only when accompanied by a focal ocular reaction. The employment of minimal doses of tuberculin is a satisfactory method of treatment for these forms of ocular tuberculosis. The acuteness of the lesion, although not a contraindication, requires prudent dosage. Success depends on an opportune commencement of the treatment, which must be long to prevent relapses. He has had no beneficial results in typical tuberculous lesions of the conjunctiva.

C. E. FINLAY.

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(Concluded from p. 789)

CORRECTION OF AMETROPIA WITH ZEISS' GROUND CONTACT GLASSES. DR. H. HARTINGER, Jena.

Following Heine's (Kiel) suggestion and directions the Zeiss ground contact lenses have been made in a test case series of thirty-nine with a corneal inner radius of from 5 to 11 mm. with intervals of 0.5 mm. and scleral radii of 11, 12 and 13 mm. The glasses for the patient have to be ground to order. As these contact test lenses in their corneal portion represent an afocal or nonrefracting system, the correction of ametropia is effected by the layer of fluid collecting between the back of the contact lens and the surface of the eye, viz., tears or physiologic solution of sodium chloride. The optical effect of the cornea is eliminated almost completely as the fluid which coats it has almost exactly the same index of refraction. In this case, full correction is attained when the virtual (bildseitige) focal point of the fluid-lens coincides with the far point of the eye. Hence, corneal astigmatism is automatically excluded almost entirely. Furthermore, as the contact glass follows all motions of the eye, all the usual optical defects disappear which are due to the combination of an immovable lens and, at some distance from it, a rotating globe, e. g., astigmatism of oblique pencils, colored difference of inclination (Neigungs Differenz), limitation of the field of vision, alteration of external accommodation effect, discrepancies in the angle of fixation in anisometropic eyes. For myopes there is the additional advantage that the retinal image is greatly magnified. In myopia of 20 diopters this amounts to 50 per cent. Additional refraction correction is necessitated by the variation in human eyes, and the suitable glass to be ground is found by means of a table of graphs made on the basis of a rectangular system of coordinates with the corneal radii (from 5 to 11 mm.) as abscissae and the refraction values of the principal focal points as ordinates. The choice of a correcting lens can also be determined by means of any ophthalmometer that, according to Heine, allows the radius of corneal curvature and the corresponding refractive power of the corneal system to be read off directly. (A projection demonstration was given of a double scale, expressing the results in diopters by means of the factor $n = 1.332$, corresponding to the Gullstrand schematic eye.) The graphic representations used are all calculated for an extremely thin fluid lens layer of fluid between the contact glass and

the cornea. The resulting errors are so small as to be practically negligible. The correctness of the graphic determination may be checked up, however, by further subjective and objective determination of refraction. In order to fit the lens perfectly to the surface of the globe, it is occasionally necessary to order one that differs from the full correction, and a correspondingly stronger supplementary refraction power.

DISCUSSION

DR. VON CSAPODY, Budapest, Hungary: Using paraffin with a low melting point it is possible to get accurate molds of the surface of the globe which can be used as models of the cornea and adjacent sclera so that the optician can turn out a perfectly fitting contact lens. To pour the wax a form is needed, a glass tube that keeps the lids apart and that is of use in removing and finishing the cast. As the paraffin changes its shape easily and quickly, one must harden it immediately before the cast is removed from the surface of the globe, by pouring iced paraffin oil (liquid petrolatum) into the glass tube. The taking of an eye model is harmless, not annoying to the patient and, with a little manual dexterity, easily accomplished. The preparation of the paraffin "negative" and its casting in plaster or metal requires more skill and is really a matter for an expert technician. Practical experience will decide which eyes can be fitted directly from the contact glass test case, and which need to be modeled (demonstration).

DR. LOEHLEIN, Jena: We all know that keratoconus of high degree can be corrected by contact lenses as in no other way, that these lenses can be worn for years without causing any trouble if taken out at night, and that they are the only means by which these patients can follow gainful occupations. It is to be supposed, *a priori,* that in the same way patients with extreme degrees of ametropia, especially high myopia with astigmatism, would obtain the best optical correction from, and would be able to wear, properly ground contact glasses. The posterior surface of such lenses could be more easily fitted to a spherical or slightly astigmatic cornea than to a conical one. Our small series of cases supports this view. It is not probable that the use of contact lenses will become as common as Heine predicts. Fick (*München. med. Wchnschr.*) claimed priority, but August Mueller (Kiel, 1889) independently worked out the entire question of what he called corneal-lenses and collected clinical data. He himself had myopia of 14 diopters. In his "Doktor Arbeit," he sharply criticized the biconcave spectacle-lenses then in use. He insisted instead on periscopic lenses made from his own calculations, and proceeded to show that the ideal optical correction of any error of refraction must consist of an extremely thin glass ground so that its posterior surface would fit accurately to the curve of the surface of the eye and move with it in all directions without being displaced. The correction of ametropia was to be effected by the curve ground on the anterior surface of the contact lens. Such contact glasses were actually ground for him, forty years ago, by Himmler (Berlin). He was able to correct his own myopia to within 1.5 diopters with a glass of this sort, but could not stand the pressure of the lens on the eye for more than half an hour. He correctly ascribed this annoyance, not to contact of the lens and the cornea, but to pressure of the edge of the lens against the conjunctival capillaries and consequent squeezing

off (Abklemmung). He tried in various ways to remedy this defect by changing the grinding of the lenses but obtained no satisfactory results. Meanwhile the expensive experiments had used up his means.

DR. LINDNER, Vienna: One must not prescribe the lenses which give the best correction but those which are best borne. We had a case of keratoconus, in which contact glasses gave a vision of 6/6; the patient could not stand them, however, although they fitted perfectly, and although she tried persistently for six months. Mueller's contact lenses, which are optically inferior, are worn with much more comfort. Zeiss' lens may fit so well that they can be removed only with great difficulty. Such lenses would not be tolerated, and of course they do not correct lenticular astigmatism. (? P. H. F.).

DR. STOCK, Tübingen: Few of us have had a chance to give these Zeiss contact lenses a practical test as they are still a monopoly of one or two firms. I myself have worn such lenses (from 1912 to 1914), prescribed for me by Professor Henker of the Zeiss firm. These were accurately determined and carefully fitted, and the optical effect was excellent. After from two to four hours, however, they caused such pain that it was impossible to wear them any longer. So far, we do not know whether, and to what extent, these lenses may actually be dangerous or injurious to the eye, and it may be our duty and to our interest, in view of possible later litigation, to warn our patients that they are wearing the lenses at their own risk. Let us hope it will be feasible to turn out less perfectly fitted ground lenses, which will be worn more easily and yet be optically satisfactory.

DR. ERGGELET, Jena: The manifest optical advantages of the contact lenses led me, years ago, to study the cause and prevention of the irritation induced by them. I experimented on my own eyes and was greatly aided by the Zeiss firm. Lenses of quartz were just as irritating as those of the usual glass. Physiologic solution of sodium chloride used as a filling fluid, back of the contact lens, is soon replaced by tears, as shown by the rapid disappearance of fluorescein coloring, and, besides, it causes no irritation. The form of the lens is important. The old lenses of Mueller which were blown, not ground, are worn with comfort for hours by almost every one. In this method optical perfection is a matter of chance, compared with ground lenses. The good points of both might be gained by combining the method of manufacture. Mueller in Wiesbaden has done this, and had his blown lens ground over by Zeiss with excellent results. Unfortunately, these blown lenses are under great tension and burst very easily; almost all of them went to pieces as soon as they were put on the grinding tool. This may have been due to two kinds of glass having been used for the corneal and the scleral portion, respectively, or to improper cooling. Discomfort is generally caused by uneven pressure and compression of vessels near the cornea. This may be relieved by regrinding the lens at the defective point. A correct fit of the scleral portion is essential for comfortable wear. This may necessitate a slightly irregular form of lens curve. This can be induced by blowing, but is practically impossible by grinding, methods. Then, too, the area of the blown lens is greater than that of the ground lens, and the pressure per unit of surface correspondingly less with equal weights. It is essential to have lenses

ground to fit each eye, as the curve of the surface, aside from the cornea, is not uniformly spherical. Direct pressure on the cornea may also cause pain and visible lesions in the epithelium, gradually developing opacities and "wreaths of pearls" and rapidly evolving alterations of tension in the corneal stroma or framework resulting from pressure, as indicated by the appearance of dark lines.

DR. VON HIPPEL, Göttingen: The physicist Wigand, then in Halle, now in Hamburg, carried out an extended series of investigations, in collaboration with Professor Henker (Jena) and used zellon (celluloid?) as well as glass; even then, the lenses could not be worn for more than four or five hours, in the best of cases, on account of marked irritation.

DR. WISSMANN, Wiesbaden: Almost invariably I had excellent results in a large number of cases of ametropia corrected by means of Mueller's contact shells. They were worn without discomfort and could be worn up to twelve or fourteen hours a day, causing no damage whatever. Although the optical question presents certain problems and the manufacture some technical difficulties, and although careful fitting is not at all simple, these sources of error are of secondary importance compared with the advantages of using these lenses with comfort and without danger.

DR. CLAUSEN, Halle: Zellon shells give excellent optical results, but the more closely and accurately they fit the surface of the globe, the worse they are borne; they can generally be worn for only a few hours. Loose fits are much more comfortable. In other cases with accurate conformity of the shells to the curvature of the globe, there was a marked suction action, causing interference with circulation and resulting in nutritional disturbances indicated by numerous small erosions of the corneal surface. If the shells are freely movable on the surface of the globe, they are usually most comfortable, but the cosmetic effect then leaves much to be desired.

DR. HARTINGER: It is too early to decide as to the respective merits of blown (Mueller, Wiesbaden) and of ground (Zeiss, Jena) contact lenses. Loosely fitting shells were more comfortable at a time when there were only three or four available forms of Zeiss' glasses, but now there are thirty-nine. If the best optically ground lens is not comfortable and one has to prescribe a less perfect correction, the lens can always be brought up to optical perfection by alteration of the curvature of (to?) the corneal surface.

PHOTOKERATOSCOPY. DR. MARC AMSLER, Lausanne, Switzerland.

The geometric form and histologic structure of the cornea are of practical importance in these days of industrial insurance and occupational ophthalmology. The Placido disk, now fifty years old, is still invaluable and shows the slightest deviation from the normal in curve, transparency or regularity of surface. The only reason why the scientific and practical harvest of observations with this instrument is so exceedingly small is that the pictures could not possibly be faithfully copied offhand or recorded in accurate and descriptive terms. Photography, alone, can give an objective document which one can make use

of. At my suggestion and with the technical details supplied by their Dr. Hartinger, the firm of Zeiss has put out an apparatus that accomplishes this in a satisfactory manner. Accurate focusing is simple, and there are various disks that can be used for one or another case.

ETIOLOGY OF KERATITIS DISCIFORMIS. DR. GRUETER, Marburg.

Eighteen early cases, dating from 1915, were selected from a large material. In fifteen of these cases there was herpes infection, in one case, vaccine (cowpox) infection, and in two, varicella. Inoculation of rabbit's cornea in herpetic keratitis disciformis gave negative results in two thirds of the cases for the following reasons: 1. In late cases the vesicles had dried up and the virus had become too attenuated, or the vesicles themselves were the result not of living pathogenic micro-organisms, but of "dead" toxins. 2. The content of the vesicles in keratitis disciformis, as in all other forms of vesicular corneal disease on a herpetic basis, is of slight virulence as compared with the high toxicity of the fresh material of keratitis dendritica. Occasionally (two cases) an unusually neurotropic form of herpes is found with slight tendency to epitheliotropy which, on inoculation into the cornea, produces little if any inflammatory reaction. This keratitis, however, mild as it was, was followed by a severe and rapidly fatal encephalitis. The use of brain material from these cases gave unequivocal proof of the biologic identity of the material derived from the vesicles of keratitis disciformis with the virus of typical herpes.

CLINICAL DIFFERENTIATION OF VARIOUS FORMS OF HERPES CORNEAE. DR. H. PFLIMLIN, Basle, Switzerland.

In tests of corneal sensitivity with the hair apparatus of von Frey, the normal cornea shows a threshold value of from 5 to 10 Gm. per square millimeter, for recognition of contact, and from 10 to 20 Gm. per square millimeter for sensation of pain. Three types of herpes can be distinguished: 1. Herpes febrilis, in which there is a history of sickness with elevation of temperature. Sensibility is unchanged in the unaffected eye, and is lowered slightly if at all in the intact portions of the herpetic cornea. 2. Constitutional herpes. In this condition both corneae show diminution of sensibility, which is most marked, of course, in the diseased area. The previous history is not significant. To determine whether the hypesthesia is a result or a cause of the herpes, 400 normal eyes were tested. About 10 per cent showed a constitutional hypesthesia of the cornea. In 3 cases it had been inherited from the maternal parent. This condition corresponds to the herpes preparedness (Bereitschaft) of Doerr. 3. Traumatic herpes, in which there is a typical history of injury, without any febrile manifestations. Sensitivity is about as in herpes febrilis; hence, constitutional hypesthesia is not a predisposing factor in these cases. The slit-lamp shows no distinctive features in these three forms. All have about the same clinical course, which is aborted if tincture of iodine is applied during the first eight days. In protracted, neglected cases the course is much slower in constitutional herpes than in the other forms. Animal experiments must decide whether these three clinical forms are due to the same, or to three distinct, varieties of virus.

DISCUSSION

DR. GRUETER, Marburg: There must be a special local or general susceptibility (disposition). Not every one gets herpes. In the positive cases of inoculation of rabbit keratitis in a human cornea, the vaccination-effect was noted only if trigeminus sensitivity had previously been excluded by the deep orbital injection of alcohol. Injury alone, therefore, is not sufficient to make material, which is of itself virulent, "take hold" and cause infection. Still there is a traumatic herpes which is demonstrated in those cases in which in apparently normal but really specially disposed and susceptible eyes corneal herpes develops a day or two after injury of the cornea.

EXPERIMENTAL RESEARCH ON ANAPHYLAXIS. DR. W. RIEHM, Würzburg.

1. Animal experiments show that under special conditions trauma is apt to localize general processes of hypersensitivity at the point where it takes effect (special instance of nonspecific influence in the positive sense). 2. Albinos react quite differently from pigmented animals in experiments on sensitization. Local anaphylactic inflammation, to begin with, is much milder and runs a much more rapid course in albinos, but it is more difficult to check the anaphylactic poison (noxae) in the tissues. On the other hand, they are more subject to shock even when treatment is locally successful, and they show the phenomenon of elective sensitization. Hence, this can have nothing to do with organ-specificity of the uveal pigment. 3. By elective sensitization I mean merely that the tissue in question is more highly sensitized than the rest of the organisms, so that it is fitted to take up the greater part of the circulating antigen from the blood. It is not surprising that histologic changes are found in other tissues, too. Besides, in case there is an oversupply of antigen, the entire reticulo-endothelial apparatus of the organism is mobilized and, under certain conditions, there may be a clinically demonstrable inflammation, e. g., of the eye, even if there has been preliminary treatment from another point of the body. The laws of elective sensitization, of nonspecific influencing (Beeinflussung) and of more rapid reactivity of the integument point the way in the organism not only to lifeless antigen but to living antigen, i. e., bacteria, as well. The organism itself therefore determines in these cases the point at which fixation (Abbindung) and elimination are to take place, and the corresponding three laws of metastasizing are determined by reactions analogous to immune processes, except that here agglutinins which are to a certain extent limited to and peculiar to the cell (Zellstaendig) take up the bacteria from the blood and neutralize them.

DISCUSSION

DR. A. VON SZILY, Münster: It has not been proved that the effect of a single injection has anything to do with anaphylaxis. The immunobiologists (Uhlenhuth, Friedberger, et al.) claim that in a previously untreated animal, any reaction under these conditions is a primary-toxic process which is in no way related to true anaphylaxis.

FOCAL OCULAR REACTIONS AFTER EXPERIMENTAL LESIONS OF CAPILLARY ENDOTHELIA. DR. A. VON SZILY, Münster.

Various irritating substances, such as pyrifer, tuberculin, homologous and heterologous serums, homologous organ-extracts and blood, were used. Wherever applied and when administered parenterally, even in small doses, all these agents produced inflammatory distance-reactions in the intact (unberührt) eye as well as in other organs (lung, liver, brain). The intra-ocular lesions observed were mostly in the nature of dilatation of vessels, edema of the ciliary body and large or small accumulations of lymphocytes in all parts of the uveal tract. It seems that many toxic substances, some of which are produced in the organism itself, especially in that of rabbits, act selectively on the capillaries. The shaken-up blood, especially that of pregnant animals, is particularly active as an early poison ("Frühgift," Freund). Some experimental animals die after the reinjection of minimal quantities of their own blood. The decomposition (abbau) products of intermediate metabolism which form with coagulation of auto-blood are separable (abtrennbar) from histamine, and of well known and characterized pharmacologic action (Freund, Zipf and Wagenfeld). They are the actual carriers of the irritative action. These focal reactions in the reticulo-endothelial apparatus are also of practical importance in the study of the not uncommon cases of "endogenous" uveitis of indeterminate etiology. Products of the individual organism may break through the provisions of the law of autotoxic immunity and give rise to conditions which are noted generally in the human organism, too, only as a result of parenteral albumin-decomposition.

DISCUSSION

DR. MARCHESANI, Munich: Focal reactions can easily be brought about which are limited to the uvea of one eye, when an infection with slightly virulent germs is started from the other. What is the serologic explanation of this selective localization?

DR. KRUECKMANN: It is possible that in phlyctenular processes decomposition products play a rôle, which are formed in the organism but which are not strictly due to tuberculosis-metabolism.

DR. FREUND, Münster: The formation of chemical substances, which have an action like that of capillary poisons originating in the organism, is obviously a general form of reaction to various noxae and injuries which affect the body. The circulation of these matters in the blood will particularly affect those capillary areas which are under local inflammatory damage, as in a tuberculous focus. The focal reactions can therefore be explained as the combined action of the local inflammatory irritation and that of the capillary poisons which have developed indirectly (intermediaer).

DR. VON SZILY: Even a nonspecific irritation may act at a point of least resistance and influence the eruption of a phlyctenule, and vice versa (sic). The eye is in every respect to be considered as part of the entire organism. Hitherto, no relation of one eye to the other which did not fit in with this conception has ever been shown by unequivocal experimental data. Moreover, from the eye itself exactly the same reactions can be set in motion in the organism as in any other kind

of influence, and the participation of the fellow eye takes place entirely within the confines of accepted laws and tissue conditions and their relation with the organism as a whole.

SPECTROGRAPHIC STUDY OF LIGHT ABSORPTION BY THE HUMAN LENS. DR. M. BUECKLERS, Berlin.

Cataractous lenses of patients, aged from 48 to 84, were the material. As the capsule had generally been opened and some faintly colored cortex lost, the absorption spectra (shown) represented minimal values. Spalteholtz' clearing fluid was used to increase greatly the transparency without altering the color. The lenses were carefully fastened between plane parallel plates of glass and examined, by nitra-light of the slit-lamp, with Zeiss' small spectrograph for visible light. Black and white and color photography shows that yellow lenses absorb violet; dark yellow to light brown lenses, violet and blue; dark brown lenses, violet, blue and also almost the entire green area of the spectrum. Photomicrographic studies of the continuous spectrum threw no light on the nature of the yellow pigment, and allowed no definite conclusions as to the validity of colors (? Farbentüchtigkeit) before operation although they showed clearly how variously color perception is influenced by the chromatic conditions of the lens.

RETINAL CHANGES AFTER IRRADIATION. DR. HOFFMANN, Königsberg.

The effect of "hard" roentgen rays is rarely noted, and for that reason has been denied. Two cases of injury were reported: In one, there was endothelioma of the orbit, and in the other, cancer of the lid, going on to destruction of the eye and necessitating exenteration and removal, respectively. Histologic examination showed dilatation of the retinal and choroidal capillaries and veins, and contraction of the arteries. No decided changes occurred in the vessel walls. Degeneration of the pigment epithelium and changes in the ganglion cells, fibrils and glia were seen. These lesions were probably due to contamination by "soft" roentgen rays and by diffuse radiation (Streustrahlung) which produce just such changes. In rabbits, irradiation also causes loss and bleaching of hair, and, on repeated exposure, conjunctivitis, cataract and retinal and choroidal lesions.

EXPERIMENTAL CATARACT DUE TO SMALL DOSES OF ROENTGEN RAYS. DR. ROHRSCHNEIDER, Berlin.

Animal experiments prove that of all ocular tissues the lens is the most sensitive to roentgen rays. In rabbits its sensitiveness equals that of the hair papillae in the skin; that is to say, the "epilation" dose, administered once, produces cataract. If this dose is divided and administered at intervals of two or three weeks, cataract still develops, while the hair does not seem affected by this divided dose and does not react until the total dose has been increased to about double that of the epilation dose. Evidently, there is an accumulation of these divided doses in their action on the lens, so that a lesion can be caused by the repeated application of doses each of which is smaller than the cataract dose, if this is exceeded in amount by the total dose.

THE ACTION OF IRRADIATION ON THE EYE. DR. ERGGELET, Jena.

In operatives in the manufacture of x-ray tubes, lesions are noted, in the course of years, in the skin of the lid, the brows, lashes and bulbar conjunctiva, which do not become extreme; they are followed, finally, by changes in the lens, which progress. They are probably due to frequent nonuse of the protecting screen or disk in front of the observation window, and as the weighting (Belastung) of the tubes varies in the course of manufacture, it is impossible to determine accurately either the intensity of irradiation or the category of the rays involved. Rabbit's eyes were tested with Bucky's "Grenz" or borderline rays. Prolonged irradiation failed to produce opacities of the lens, but sufficiently intense irradiation produced corneal epithelial changes, and in pigmented animals there was an inundation of the cornea with pigment from several points of the marginal ring like that reported and described by Lochlein as caused by thorium X. Further experiments with soft rays increased the probability that the lesions in workers on x-ray tubes are due to repeated sharp, short blows with x-rays, rather than to any one kind of ray. The data of occupational arrangement supports this view. Probably the lens has a "ray pessimum" of species, intensity and frequency in the time unit as compared with other ocular tissues.

DISCUSSION

DR. VOGT, Zürich, Switzerland: The senile lens, becoming yellow or yellowish red, absorbs not only ultraviolet and violet, but blue and green as well. Hence, a dioptric, not a retinal, blue-blindness occurs, as noted at least sixty years ago by Liebreich who found evidence of disturbance of color vision in the paintings of aging artists (Turner, P. H. F.) and interpreted them correctly. Such persons are also (dioptrically) yellow-blind, as I have found in subjects with a yellow or reddish-yellow lens. This is analogous to the disturbance of yellow sensation produced by electric light and, to a still greater degree, by kerosene or candle light. Another and quite different form of acquired alteration of color sensation is the chromatic tuning up or down (Umstimmung) by light (Tschermak). This effect is shown instructively in paintings. In a snow scene in bright light, all the tones, direct as well as reflected, are warm and from the red end of the visible spectrum. In a sunless scene, the cold hues from the blue end come into play. Erythropia due to dazzling is another expression of this alteration of chromatic temperament due to retinal and not to dioptric reactions. Small doses of roentgen rays cause in rabbits, at first, small cell clouding of the fluid of the anterior chamber with exudates on the lens capsule and iris, both transitory conditions, and much later, after 168 days of irradiation, changes in the posterior (axial) cortex of the lens. The great sensitivity of the lens suggests extreme caution in therapeutic irradiation.

DR. KLEIBER: Guinea-pigs treated with more than 800 R units died before changes were noted in the lens. Lower doses, 250 R and more, caused the evident formation of cataract without phenomena of epilation in the course of several months after a single irradiation; the cataract was of the same form as the well known roentgen opacity in rabbits.

DR. BLAICKNER, Salzburg: Biologic effects of irradiation suggest some difference between soft and hard rays, especially the very hard rays near the gamma rays. These may be less injurious, a matter of practical importance for clinicians who are in the fortunate position of being able to choose between roentgen rays and radium in the treatment for ocular diseases.

DR. SCHEERER, Tübingen: Clinical experience supports the theory of the embryologic origin of the lens as a derivate of the integument with special characteristics determined by its special features of metabolism. In ocular tuberculosis we never give more than 80 per cent of the unit skin dose in the course of a year. When this is not feasible, as in the case of tumors, and when the eye cannot be protected, we expect cataract to form within two years. Extraction in the capsule, however, gives excellent results in these cases.

DR. COLMBERG, Berlin: I cannot agree with these conclusions. In spite of the epithelial (ectodermal) nature and origin of the lens, it is not as easily damaged by roentgen rays as the epithelial skin. One must not forget that there is a decided difference in the action of radiant energy on the two forms of epithelium. In the case of the lens, the lesion does not develop for a number of months, or even years. A delayed action of this kind is not seen in any other living tissue. An explanation may be given by the fact that the lens, enclosed in its capsule, is an organ with very slow metabolism. Possibly the posterior capsule, without any row of cells, is the main metabolic filter. As a result of roentgen injury to the fibers (? P. H. F.) contained in the lens capsule, a surplus (Anreicherung) of decomposition and metabolism products may take place just at the posterior pole under the capsule and in the sutures of the lens-star. Constant shifting of the contents of the lens under the influence of accommodative change of form may also contribute mechanically to the transportation of this matter. The decomposition (abbau) products may act at the point of greater concentration similarly to some of the agents mentioned by von Szily in the preceding paper. Cataract from irradiation by heat, light or radium may be, accordingly, a form of focal reaction peculiar to the lens.

DR. THIES, Dessau: In a case observed in 1909, severe injury resulted from welding for several weeks with the eyes unprotected. In the right eye the severest changes occurred at the macula, with vitreous opacities. Improvement followed. Very slight changes were also found at the macula of the left eye. The compensation commission decided that the lesions in the right eye were due to injury, but disregarded those in the left. Vision in the left eye gradually failed (perception of fingers at from 4 to 5 meters); in 1928, it was found that the macular lesions had steadily advanced. The connection of changes in both eyes with lesions produced by radiation in 1909 was proved positively by the discovery, by dilation of the pupil, of changes in the anterior lens capsule. These must always be looked for in injuries caused by radiation.

DR. HESSBERG, Essen: In early roentgen treatment of an eye seriously threatened with acute or sluggish (schleichend) iridocyclitis, particularly after injury, treatment which is generally successful, one must

weigh the possibility of producing a cataract against the hope of preserving the form of the eye and possibly some vision, later on, in a favorable case. This is the lesser of two evils, particularly as the extraction of such roentgen cataracts in the capsule gives excellent results in these as in other, particularly the tuberculous, forms of slowly progressive iridocyclitis. This also applies to irradiation of eyes with hemorrhagic glaucoma, which otherwise are doomed to destruction. Radiation is applied, refracta dosi, in an amount less than 80 per cent of the skin unit dose without much danger of cataract formation.

DR. ROHRSCHNEIDER: In industrial injury by roentgen rays one should speak of damage, in a medicolegal sense, only when irreparable changes which interfere with function have taken place in the eye. There may be differences in sensitivity to roentgen rays between various species, as there is, for example, between the human being and the rabbit. The hard roentgen rays have proved less injurious as they have a greater therapeutic breadth.

THE ACTION OF STRYCHNINE NITRATE ON SOME OCULAR REFLEXES. DR. KYRIELEIS, Würzburg.

The subcutaneous injection of from $\frac{1}{20}$ to $\frac{1}{12}$ grain (from 3 to 5 mg.) of the drug caused the light reflex of the pupil to take place in jerks and to reach its contraction maximum more quickly, as shown objectively by curves taken with the kinematographic method of Loewenstein (Bonn). The latent period of caloric nystagmus was shortened, on an average, 44.14 per cent in twenty-six cases, in which the cold test with minimal irritation (Kobrak) was used. Optomotor nystagmus showed an increase, averaging more than 10 per cent, in the frequency of twitching as registered with the lever nystagmograph. Dark adaptation was accelerated, and maximum sensibility, accordingly, reached more quickly, but the previously noted maximum was never exceeded.

FATTY DEGENERATION OF THE RETINA IN ENDOPHTHALMITIS AND SYMPATHETIC OPHTHALMIA. DR. P. A. JAENSCH, Breslau.

After injury to the retina by toxins (albuminuric retinitis) sudan-positive substances in small to medium-sized droplet form are found in Mueller's supporting cells, particularly in the basal plates, in the nuclear region of the neuroglia among the internal nuclei, and in the degenerating rods and cones. After penetrating injury of the eye, pus and fat cells invade the retina from in front and spread through this tissue. Fiber baskets (Faserkoerbe) were found in these cases, in nephritic retinitis, in sympathetic ophthalmia and in iridocyclitis following an operation for glaucoma in a patient with nephrosclerosis. Pigment epithelial cells may migrate into the tissue of the retina which has undergone glia degeneration and, rather infrequently, fatty degeneration. The intensity of this fatty degeneration of the retina depends on the course of the intra-ocular infection, on the virulence of the bacteria which have penetrated the globe, on special characteristics of the toxins, and on the length of time during which these damaging factors have acted on the retina.

DISCUSSION

DR. MARCHESANI, Munich: In the central nervous system and retina, the microglia cells (Hortega) are the first to undergo fatty degeneration. Epithelial cells that have lost their pigment may migrate into the retina and assume strange forms with pseudopods or, later, long processes, so that they simulate Hortega cells and suggest that, even late in life, microglia cells may develop out of the extremely productive and fruitful cells of the pigment epithelium.

THE HISTOLOGIC STUDY OF RETINAL GLIOMA. DR. MARCHESANI, Munich.

Eleven gliomas examined with the newer glia stains showed a uniform structure. The main mass was formed of primitive neuroepithelial cells, some of which differentiate to cylindric cells which form rosets. This may go on to the formation of cystic spaces with a lining membrane, suggesting the imitation of the neural tube or, at least, the marked tendency of neuro-epithelium to fold in on itself and form hollow organs. Embryonic glia cells with protoplasma processes are also found in the form of primitive spongioblasts, astrospongioblasts and astrospongiocytes. In the lower developmental forms, the processes of the glia cells have a markedly syncytial structure and completely fill the interspaces present. The more highly developed glia cells have long, extended processes and more or less perfectly developed vascular feet (Gefaess-fuesse). Vessels appear to be centers in relation to further, higher development. In many gliomas the ripest age is indicated by the development of primitive spongioblasts and the most perfect development is attained by compactly growing tumors with slight evidence of degeneration. One tumor presented almost normal astrocytes. Similar embryonal gliomas are also found in the brain (Bailey, Cushing).

ATYPICAL COLOBOMA IN RELATION TO MYOPIA. DR. SCHEERER, Tübingen.

Photomicrographs of eight cases of developmental defect at the entrance of the optic nerve showed the anomalous beginning of the nerve within the eye, abnormal fibrous bands at the margin of the retina and eversion of the retina under the pigment epithelium. In these, as in most cases reported, there was a more or less high degree of myopia. Hence (? P. H. F.) there must be an intimate causal relation between this malformation of the papilla and the refraction error, which generally leads to an elongated form of the globe (Langbau), but which at times may result in a colobomatous microphthalmus. In this connection it is interesting to note that highly myopic eyes may have an abnormally small corneal diameter. The fundus changes in myopic eyes are due to malformations of the papilla, and the secondary degenerations occurring in the course of life explain the development of progressive changes, such as conus or stretching, to the extent of posterior staphyloma which is to be regarded as an atypically located, ectatic coloboma.

DISCUSSION

DR. LAUBER, Vienna, Austria: Many eyes with congenital anomalies of the optic papilla show corneal opacities (Elschnig). In cases of

inferior, or nasal, conus, with reversed vessel distribution, etc, the traces of a previous corneal inflammation and sequelae in the form of opacities are found much more frequently than in the normal fellow eye. The same is true of anisometropia, with involvement of the more highly ametropic eye, whether hypermetropic or myopic. An eye with this condition is also much more susceptible than its fellow to various diseases, such as eczematous keratitis. In this case one probably has to deal not merely with topographic malformations but with a true biologic inferiority of the ocular tissues, and a correspondingly increased susceptibility to disease of exogenous or endogenous origin.

THERAPEUTIC STUDIES AND SUCCESS WITH AMYL NITRITE IN DISEASE OF THE FUNDUS. DR. VON IMRE, Budapest, Hungary.

Sudden spastic contraction of the retinal arteries is not the only disease of the eye in which the inhalation of a few drops of amyl nitrite often acts remarkably well. In some cases of chronic chorioretinitis of doubtful etiology, pigment-degeneration of the retina, arteriosclerotic nutritional disturbances, albuminuric retinitis, and vascular contraction in arterial hypertension, spasm of smaller vessels is part of the clinical picture and probably the actual direct cause of the lesions. Systematic hyperemization of the choroidal and retinal vessels may improve the nutrition of the endangered tissues, via the blood supply. Daily inhalations resulted in improvement of vision and enlargement of the visual field in a number of cases.

DISCUSSION

DR. IGERSHEIMER, Frankfurt: The factor of autosuggestion must, of course, be considered, but these observations are significant in view of the most recent theories of the rôle of angiospasm in albuminuric retinitis and of histologic studies in apoplexy. In addition to changes in the intima of cerebral vessels, spastic reactions in the vascular area of the ophthalmic artery played a part in a case of severe optic nerve disease which I reported in 1929 (*Ztschr. f. Augenh.* **69:** 47).

DR. BRUECKNER: French authors have laid stress on vascular spasm as an etiologic factor, particularly in toxic ambylopia. In one case, the administration of a compound sodium nitrite solution was followed by marked improvement in the appearance of a nerve head which had begun to show pallor.

DR. MYLIUS, Hamburg: I wish to warn against the too general use of vasodilating drugs. A sudden lowering of blood pressure may be dangerous, as is a gradual drop also in cases in which high blood pressure is requisite for the elimination of decomposition products (Schlacken) of metabolism. Drugs like amyl nitrite should be used only in cases in which primary vascular spasm is an immediate cause of increased blood pressure, as there is then no question of causing retention of waste products.

REGENERATION OF VITREOUS. DR. A. BRUECKNER, Basle, Switzerland.

Experiments with rabbits by Guggenheim and Franceschetti demonstrated an increase of albumin content in the vitreous. Microscopic sections show the origin of this albuminous fluid to be the ciliary processes. In the early stages, coagulated albumin can be seen extending

from the equator of the lens to the region of the posterior pole. Later, it is found in the posterior portion of the vitreous, and in the anterior and posterior chambers. The presence of numerous circumscribed detachments of the limitans interna of the retina caused by cystlike (blasenartig) structures which penetrate into the vitreous suggests the possibility of such limited replacement of fluid in the vitreous by the retina or its glia. The vessels on the disk and the flat part of the ciliary body take part in supplying fluid to the retina only if inflammatory phenomena (cell migration) are present, even in the slightest degree. The iris is not involved after puncture of either the anterior chamber or the vitreous but, as has been well known for a long time, it plays a rôle in inflammatory processes. The speaker has already shown that the corneal endothelium may become of importance biopathologically, so that all intra-ocular tissues, it appears, play a part in vital processes such as regeneration. The fact that in the absence of inflammation the ciliary processes almost exclusively serve to replace fluid of the vitreous while the retinal vessels show at most occasional hemorrhage per diapedesin but are otherwise inactive speaks for a specific function of the ciliary processes in which the ciliary eipthelium may play a rôle. The exit of fluid from the retinal or glial tissue, on the contrary, is in all probability caused quite mechanically by the lowering of intra-ocular tension, as the phenomenon is noted also, in a somewhat lesser degree, after puncture of the anterior chamber and even in the surviving (ueberlebend) eye.

Dr. Jess, Giessen: The circumscribed globular (buckelfoermig) detachments of the limitans interna described have been noted in experimental choline poisoning of rabbits. Ophthalmoscopically, they appear as small white spots suggesting nephritic or diabetic retinitis.

A Clinical Study of Two Cases of Pulsating Exophthalmos. Dr. M. Baurmann, Göttingen.

The blood pressure in the retrobulbar portion of the central retinal vein was increased, as shown by continuous observation of the venous pulsation to the same degree as in a series of cases of papilledema with prominence up to 6 diopters. Hence, this condition is not due alone to increased pressure in the vein, as required by Deyl's theory, supported by Dupuy-Dutemps, of the genesis of choked disk. In fact, in a number of cases of manifest papilledema the tension was decidedly lower than in the two cases of pulsating exophthalmos.

Iridencleisis Operations. Dr. Blaickner, Salzburg.

From 1926 to 1930, fifty-six operations were performed, all but one for simple or chronic inflammatory glaucoma. The complications were: severe hemorhage in two cases, injury to the capsule, with cataract, in three cases (changes in the lens previously noted in two) and purulent iridocyclitis necessitating enucleation in one case. The probability of infection incidence is similar to that in cataract extraction, as the compressed portion of iris remains in situ while in all other glaucoma operations it is excised. Vision was improved in seventeen and unaltered in thirty cases; it became worse in five, and was lost in three (one was the case of infection). The two other patients had defective fields and

only a remnant of central vision before operation. Tension was "primarily" reduced to normal in 78 per cent. The visual field was improved or maintained in 66 per cent. The technic was as follows: A broad, thick conjunctival flap (Schieck) was made, 1.5 cm. from the limbus, and Tenon's capsule opened as much as possible to prevent early and late infection of a severe nature as well as the mild (banal) forms which cause merely a slight fibrinous exudation into the stroma of the iris and interfere with the carpeting (Auskleidung) of the wound canal with pigment epithelium of the iris. These were Holth's original ideas. Hence, miotics are not used shortly before and after operation, or during the entire period of healing. A keratome incision is made, as peripheral as possible, 2 mm. from the base of the conjunctival flap. If an iridodialysis should result, no harm is done. The conjunctiva is always sutured. In this way, from the time of the operation, instead of a valve-fistula (Ventilfistel) there is established a sort of sluiceway (Schleusenanlage) which permits a permanent slitlike permeability of the wound canal. The peripheral site of the keratome incision and the meridional iridotomy have as an object not only the fistulizing effect of iridencleisis but the additional factor of freeing (lueftung) the periphery of the posterior chamber and the avoidance of synechiae of the ciliary portion of the pigment epithelium of the iris with the ciliary processes and the thickened zonule. Operations involving an incision of the angle of the anterior chamber probably act by a zonulotomy. In two of my cases an irido-zonulodialysis and an irido-zonulectomy, respectively, brought about complete recovery. Generally, the iridencleisis suffices, by producing an iridotasis and possibly a sort of zonulotasis, to allow a free flow of endocular lymph from the point of production to the anterior chamber or the operative fistula. Contraindications are met with in juvenile glaucoma, glaucoma complicated by cataract and subluxated lens (Schlotterlinse).

CONTRACTION OF THE CONJUNCTIVA IN THE LID FISSURE. DR. REITSCH, Hirschberg.

Even in incipient pterygium the semilunar fold is pulled forward to the surface of the globe. In normal eyes this is prevented by a special mechanism which one best recognizes by noting the motion of the caruncle and semilunar fold accompanying rotation of the globe. This suggests that a shortening of the conjunctiva in the area of the lid fissure is inadvisable. The impression of a redundant bulbar conjunctiva is caused in advancement operations by a simultaneous advancement of the "nasal fornix." This shortening of the conjunctiva bulbi can be prevented by reestablishing the status quo ante of the lateral relations of the fornix by a fornico-conjunctival fixation at the proper topographic-anatomic point.

EXPLOITATION OF THE BLIND AND OF SUFFERERS WITH OCULAR DISEASE BY QUACKS, ETC. DR. FLEISCHER, Erlangen.

After adjournment of the meeting, Dr. Fleischer announced that, at the request of the blind, he would read a paper with the foregoing title before the German Congress of the Blind, Nurenberg, at the end of July. He requested that information on this topic be sent to him.

OXFORD OPHTHALMOLOGIC CONGRESS

DERRICK T. VAIL, JR., M.D., *Reporter*

Twentieth Annual Meeting, July 10-12, 1930

DR. A. BERNARD CRIDLAND, *Master,* in the Chair

Most of the members resided in Keble College during the three days, and this feature, as well as the splendid program, combined to make a most enjoyable and valuable occasion. There were about 148 members present, among them overseas members, including: Dr. Patry, Geneva; Dr. Schüller, Vienna; Dr. Van Heuven, Utrecht; Dr. Gonin, Lausanne; Dr. Coppinger, India; Dr. Dick, Curran, Canada; Dr. Harry Friedenwald, Dr. Tyson, Dr. Bernard Samuels, Dr. Randall and Dr. Derrick Vail, Jr., from the United States.

After an admirable address of welcome by the Master, the congress opened on July 10, with a symposium on toxic amblyopia, including retrobulbar neuritis, introduced by Dr. H. M. Traquair of Edinburgh, dealing with the ophthalmologic point of view. His address was based on an experience with 160 cases of retrobulbar neuritis, and he inclined toward the view that nasal sinuses do not play much of a rôle in the production of the disease, only two cases out of his series being attributed to the posterior nasal sinuses. He also was skeptical about disseminated sclerosis as a cause, since in relatively few of his cases did multiple sclerosis develop after several years' observation. He advanced the theory that retrobulbar optic neuritis may be a disease of the nerve similar to multiple sclerosis but not disseminated. He discussed the great importance of careful field studies, and he showed many slides from his cases emphasizing his point, particularly the detection of quadrantic or hemianopic features in the scotoma, which would locate the process at the chiasma. Dr. Traquair then discussed toxic amblyopia, mentioning tobacco poisoning particularly.

Sir Farquhar Buzzard, Oxford regius professor of physic, gave the next paper on this subject from the standpoint of a physician. He objected to the diagnostic term retrobulbar neuritis, and stated the belief that all cases should be considered as due to multiple sclerosis unless proved otherwise. He quoted E. B. Dunphy's recent work to bear out his belief.

Dr. J. A. Gunn, professor of pharmacology at the University of Oxford, contributed a valuable paper on the pharmacologic aspect of this subject, limiting his remarks to toxic amblyopia. He said that if one considers all drugs which produce toxic amblyopia one finds that these are nerve poisons and not vascular poisons. He mentioned his work in producing lens opacities in animals by injection of cobra venom. Among other topics he brought forth the theory that one reason why tryparsamide might be more dangerous in the presence of beginning optic atrophy due to syphilis was that it was a pentavalent arsenic compound, and these have been shown to penetrate more easily into the central nervous system than the trivalent ones such as arsphenamine.

These three papers excited a lively and somewhat controversial discussion, few favoring the nasal sinus etiology of retrobulbar neuritis.

Among the leaders in the discussion were Davies, Gray Clegg, P. H. Adams and Rosa Ford.

Dr. Bernard Samuels of New York showed many excellent lantern slides illustrating his paper on "Opacities of the Vitreous," which, unfortunately, he did not have time to read. This was a decided loss to the audience. The lantern slides, however, created great enthusiasm.

After luncheon in Keble Hall, the congress again convened and heard an address by Sir Oliver Lodge on "Modern Theories on the Nature of Light." This grand old man of science gave his talk in an informal manner, with side remarks explaining the more abstruse parts of his subject. He began with a description of the older theories, and finished with the modern or quantum theory of light.

Later in the afternoon, the members of the congress were entertained at tea in the gardens of Worcester College by the Provost and Mrs. Lys. In the evening the annual dinner was held in Keble Hall. The Master reviewed the history of the Oxford Ophthalmologic Congress, and a toast was drunk to its coming of age.

On Friday, July 11, Prof. Dr. Artur Schüller of Vienna gave an address on "Recent Developments in Radiology Associated with Ophthalmology," illustrated with lantern slides which showed normal and pathologic variations, especially of the sphenoid. Several pictures of calcified internal carotid arteries were particularly noteworthy. His paper was discussed by Dr. A. W. Bowie (London) and Mr. W. Edmund Jones (Leamington).

Dr. Harry Friedenwald, Baltimore, read a most able and colossal dissertation on "Pathologic Changes in the Retinal Blood Vessels in Arteriosclerosis and Hypertension." The complete paper will appear in the transactions of the society. This was the Doyne Memorial lecture, and as such was the chief attraction of the congress. The Master, seconded by Mr. P. H. Adams as Deputy Master, presented Dr. Friendenwald with the Doyne medal. It was particularly pleasing to the American members present to see their distinguished fellow countryman thus highly honored.

In the afternoon Professor Gonin of Lausanne reviewed his work on "Detachment of the Retina and Its Treatment." His technic of ignipuncture was described in detail, and was illustrated with slides of fundi pictures and of the method of operating. His statistics showed 60 per cent definite cures in recent cases. The enthusiasm of the members was remarkable.

The address was discussed by Mr. A. W. Ormond, Mr. Charles Goulden and Mr. Frank Juler of London; Mr. Percival Hay, Sheffield; Mr. N. C. Ridley, Leicester; Sir William Lister, of London, who showed several excellent lantern slides of photomicrographs of retinal holes, and by others. The tone of all discussions was most favorable, all speakers citing cures by Gonin's method in their own cases.

On Saturday, July 12, Dr. H. M. Traquair read a paper "Notes on New Instruments and Technic in Cataract Operations." He showed a new iris forceps, speculum, conjunctival needles and a wick to be inserted at the outer canthus to keep the conjunctival sac dry during operation. He discussed a suture of an upper lid which is to be fastened to the cheek afterward by adhesive strips.

Mr. T. Harrison Butler read a paper on "Keratoconus Posterior," which he tentatively introduced as a new disease. It is, briefly, a shortening of the radius of curvature of the posterior surface of the cornea, the anterior curvature remaining normal.

Dr. T. Louis DeCourcy of Liverpool presented an address on "Industrial Keratitis in Workmen's Compensation," the principal premise being the great importance of determining the rôle played by previous injury in exciting this disease, and the plea for an official action by the ophthalmologic body so that unanimity of opinion could be presented in court. Dr. J. A. Van Heuven of Utrecht, The Netherlands, read the report of a case of recurrent chronic uveitis.

The scientific exhibits included: (1) an orthogon test, Mr. Burdon Cooper; (2) a specimen of ciliary growth causing glaucoma, Mr. Arthur Greene; (3) a model of the field of vision, Dr. Traquair; (4) a stereoscope useful in fusion training, Mr. E. B. Alabaster; (5) a hand attachment for wing tests, and stereoscope, Mr. Ernest Maddox; (6) slit-lamp drawings of the lenticonus posterior, Mr. Harrison Butler; (7) roentgenograms of optic canals, Dr. Randall, and (8) demonstration of rabbits with transparent corneal grafts, J. W. Tudor Thomas. The last exhibit was most striking. Each of four rabbits had a corneal transplant (heterogenous) on one eye. These were successful in that the optic center was entirely clear in each case. The investigator had presented his technic before the Ophthalmologic Section of the British Medical Society in April, and this paper will shortly appear.

Book Reviews

THE ANNUAL REPORT OF THE ROYAL LONDON OPHTHALMIC (MOORFIELDS) HOSPITAL, 1929.

This report records the commencement of the first portion of the scheme of extension and reconstruction, which has recently been inaugurated. The plan includes the providing of facilities for patients of private means who are debarred from the general wards by their ability to pay something for their treatment, but who have not sufficient means to meet the expenses of a nursing home, an institution to which London surgeons refer those who are able to pay the usual fees.

This is a decided innovation, and the example set by Moorfields should be followed by similar institutions elsewhere, it being as much an act of charity to provide those of moderate means with adequate facilities as it is to care for the destitute. Additional accommodations are also provided by the hospital for the enormous outpatient service, 45,876 new outpatients having been treated in 1929, for the up-to-date physical therapy department and for a new library and museum, as well as for a much needed room for the medical school.

Evening demonstrations were given at Moorfields as early as 1864, but the first systematic teaching was inaugurated by Nettleship and Marcus Gunn in 1884. Since that time the hospital has been a great teaching school of ophthalmology, as well as a hospital providing its patients with all the developments of ophthalmic science. The courses of teaching are now well systematized, and the hospital is a school of ophthalmology recognized by the University of London and by the conjoint board for England as a place of instruction in ophthalmic surgery, and all the members of the honorary staff are recognized teachers in the university. The courses of instruction are designed to meet the requirements for candidates reading for diplomas and degrees in ophthalmology.

During the past year, 2,534 inpatients were admitted, the average number of daily patients who were resident being 104.32.

In 315 cases of senile cataract, the patients were operated on; 313 with iridectomy, 2 by the simple method; a preliminary iridectomy was done in 11 instances. Excision of the lacrimal sac was performed 83 times. The cautery was used for detachment of the retina in 6 cases. Corneoscleral trephining for glaucoma was performed 191 times, Herbert's sclerotomy 18 times, posterior scleral puncture 4 times and iridectomy for glaucoma (acute or chronic not specified) 41 times.

The general anesthetic of choice was a mixture of chloroform and ether, being employed in 177 cases, simple ether in but 25 cases and ethyl chloride and ether in 17 cases.

The present board of surgeons comprises: Sir John Herbert Parsons, R. Foster Moore, R. Affleck Greeves, F. A. Juler, C. B. Goulden, H. H. Whiting and P. G. Doyne.

The assistant surgeons are: Humphrey Neame, Miss Ida C. Mann, W. Stewart Duke-Elder, Rupert S. Scott and R. C. Davenport.

A short history of this venerable institution founded in 1804, prepared by Percy Fleming, accompanies the report.

W. C. P.

Obituary

DERRICK T. VAIL, M.D.

1864-1930

Derrick T. Vail, ophthalmologist of Cincinnati and well known throughout the United States and England, died on Oct. 29, 1930. Dr. Vail was clinical professor of ophthalmology in the Laura Memorial Medical College, and also clinical professor in the Miami Medical College, which merged in 1909 with the Ohio Medical College to form the College of Medicine of the University of Cincinnati. At this time he was appointed chief of the eye clinics and clinical professor of ophthalmology, a post which he held until 1912, when he retired to become professor emeritus.

He served on the visiting staff of the Cincinnati Municipal Hospital as ophthalmologist, and continued in the new Cincinnati General Hospital until he became professor emeritus in 1912. He was a founder of the American Academy of Ophthalmology and Oto-Laryngology, of which he was president in 1908. He was also a member of the Oxford Congress, the American College of Surgeons, the American Ophthalmological Society, the Section on Ophthalmology of the American Medical Association, and various other societies.

Dr. Vail was associated with Dr. Christian Holmes for ten years, following which he established for himself one of the largest practices in the Middle West, in which he was assisted in recent years by his two sons, Harris and Derrick, both of whom received their early training at Harvard and at the Massachusetts Eye and Ear Infirmary. Dr. Vail was a skilful operator and consultant. He became interested at an early date in Smith's method of intracapsular extraction of cataract, made several trips to India to study this subject, and wrote a number of papers on it. He was also much interested in detachment of the retina, and he was the first to describe a case of tularemia in man.

Owing to a disease of the heart, he was forced to retire in 1928. He lived on his beautiful estate near Holland, Michigan.

Directory of Ophthalmologic Societies *

FOREIGN

Oxford Ophthalmological Congress

Master: Dr. A. B. Cridland, Salisbury House, Chapel Ash, Wolverhampton, England.
Secretary: Dr. C. G. Russ-Wood, 12 St. John's Hill, Shrewsbury, England.

NATIONAL

American Medical Association, Section on Ophthalmology

Chairman: Dr. George F. Suker, 25 E. Washington St., Chicago.
Secretary: Dr. William C. Finnoff, Imperial Bldg., Denver.
Place: Philadelphia. Time: June 8-12, 1931.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. William H. Wilder, 122 S. Michigan Ave., Chicago.
President-Elect: Dr. J. F. Barnhill, Miami Beach, Fla.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.

American Ophthalmological Society

President: Dr. Arnold Knapp, 10 E. 54th St., New York.
Secretary: Dr. Emory Hill, Professional Bldg., Richmond, Va.
Place: Asheville, N. C. Time: June, 1931.

SECTIONAL

New England Ophthalmological Society

President: Dr. S. Judd Beach, 704 Congress St., Portland, Me.
Secretary-Treasurer: Dr. E. B. Dunphy, 520 Commonwealth Ave., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. Chester H. Bowers, 1136 W. 6th St., Los Angeles.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, Medical-Dental Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Fletcher D. Woodward, Box 162, University, Va.
Secretary: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: New Orleans. Time: November, 1931.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Southwestern Michigan Triological Society

President: Dr. Wilfrid Haughey, 65 W. Michigan Ave., Battle Creek.
Secretary: Dr. R. H. Fraser, Battle Creek Sanitarium, Battle Creek.
Time: Third Thursday of alternate months.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Donald H. O'Rourke, 217 Imperial Bldg., Denver.
Place: First floor, Metropolitan Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. Dorland Smith, 836 Myrtle Ave., Bridgeport.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

Eye, Ear, Nose and Throat Club of Georgia

President: W. L. McDougall, 50 Whitehall St., S. W., Atlanta.
Secretary-Treasurer: Dr. William O. Martin, Jr., 412 Medical Arts, Bldg., Atlanta.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: December 10 and 11, 1930.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. John C. Brown, 350 St. Peter St., St. Paul.
Secretary-Treasurer: Dr. Hendrie W. Grant, Lowry Bldg., St. Paul.

Montana Academy of Oto-Ophthalmology

President: Dr. Lee W. Smith, 125 W. Granite St., Butte.
Secretary: Dr. J. G. Parsons, Lewistown.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. B. Beeson, 232 W. De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, Valley City.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. W. McCollom, 346½ Washington St., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

President: Dr. Raymond F. Hacking, 105 Waterman St., Providence.
Secretary: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

Utah Ophthalmological Society

President: Dr. Franklin H. Raley, 9 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. H. Leroy Smith, 1005 Medical Arts Bldg., Salt Lake City.

Virginia Society of Oto-Laryngology and Ophthalmology

Secretary-Treasurer: Dr. Henry Grant Preston, Harrisonburg.
President: Dr. Fletcher D. Woodward, Box 162, University.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. S. S. Hall, Fairmont.
Secretary: Dr. G. A. Smith, Montgomery.
Place: Clarksburg. Time: 1931.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

Chairman: Dr. J. Wallace Hurff, 86 Washington St., Newark.
Secretary: Dr. Earl LeRoy Wood, 31 Lincoln Park, Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

Academy of Medicine of Toronto, Section of Ophthalmology

Chairman: Dr. Louis Joseph Sebert, 86 Bloor St., W., Toronto.
Secretary: Dr. Alexander E. MacDonald, 151 Bloor St., W., Toronto.
Time: First Monday of winter months.

Atlanta Academy of Ophthalmology and Oto-Laryngology

President: Dr. William L. McDougall, Atlantic National Bank Bldg., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., 384 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Leo J. Goldbach, 1012 N. Charles St., Baltimore.
Secretary: Dr. Angus Lloyd MacLean, Johns Hopkins Hospital, Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. John N. Evans, 23 Schermerhorn St., Brooklyn.
Secretary: Dr. Michael J. Buonaguro, 589 Lorimer St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday of February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. A. F. Luhr, 40 W. North Ave., Buffalo.
Secretary-Treasurer: Dr. Thurber Le-Win, 112 Linwood Ave., Buffalo.

Chicago Ophthalmological Society

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Secretary: Dr. Richard C. Gamble, 30 N. Michigan Ave., Chicago.
Place: Medical and Dental Arts Club. Time: Third Monday from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. William V. Mullin, 9204 Euclid Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 2064 E. 9th St., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. W. E. Burner, Guardian Bldg., Cleveland.
Secretary: Dr. M. P. Motto, Rose Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Maxwell Langdon, 1530 Locust St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Otolaryngological Society

Chairman: Dr. A. M. Hauer, 327 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Albert D. Frost, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. B. Decherd, 1717 Pacific Ave., Dallas, Texas.
Executive Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Parker Heath, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. G. G. Marshall, 122 West St., Rutland, Vt.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. W. R. Thompson, 510 Main St., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Needham, Fort Worth National Bank Bldg., Fort Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Claude C. Cody, 1306 Walker Ave., Houston, Texas.
Secretary: Dr. J. M. Robison, Eye, Ear, Nose and Throat Hospital, Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. E. L. Lingeman, 241 N. Pennsylvania St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. A. E. Eubank, Argyle Bldg., Kansas City, Mo.
Secretary: Dr. O. S. Gilliland, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Dean E. Godwin, Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Ferris Arnold, Security Bldg., Long Beach, Calif.
Place: Pacific Coast Club. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section on Ophthalmology and Otolaryngology

President: Dr. A. Ray Irvine, 727 W. 7th St., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 727 W. 7th St., Los Angeles.
Place: California Lutheran Hospital, Assembly Room. Time: 8 p. m., first Monday of each month.

Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. James A. Flynn, 1511 Rhode Island Ave., Washington.
Secretary: Dr. E. R. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Edward R. Ryan, 425 E. Water St., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 120 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. R. J. Warner, 706 Church St., Nashville, Tenn.
Secretary-Treasurer: Dr. W. W. Wilkerson, Jr., Bennie-Dillon Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

New Orleans Ophthalmological and Otological Society

President: Dr. W. A. Wagner, Pere Marquette Bldg., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. E. F. Krug, 12 W. 44th St., New York.
Secretary: Dr. William F. C. Steinbugler, 815 Park Ave., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Otolaryngological Society

President: Dr. George B. Potter, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. J. C. Davis, 425 Aquila Court, Omaha.
Place: Medical Tea Room. Time: 5:15 and 7:00 p. m., third Wednesday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit-Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. A. Vincent Walker, 833 South Ave., Rochester, N. Y.
Secretary-Treasurer: Dr. John F. Gipner, 53 Fitzhugh St., Rochester, N. Y.
Place: Rochester Medical Assn., 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Charles W. Tooker, 308 N. 6th St., St. Louis.
Secretary: Dr. Carl T. Eber, 1006 Carleton Bldg., St. Louis.
Place: St. Louis or Washington University Cafeteria.
Time: 6:30 p. m., fourth Friday of each month from October to April, inclusive.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. F. W. Sorrell, Gibbs Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. A. F. Clark, 728 Main Ave., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. A. N. Codd, 421 W. Riverside Ave., Spokane, Wash.
Secretary: Dr. Marc Anthony, Old National Bank Bldg., Spokane, Wash.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Sigurd von Christierson, 11 W. Portal Ave., San Francisco.
Secretary: Dr. Wilber F. Swett, 693 Sutter St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Third Tuesday of every month except May, June, July and December.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. Searle B. Marlow, State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. A. Harry Rubenstein, Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

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